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# DIABETES

## Its Causation and Treatment

ERNEST E. WATERS, M.D.

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# DIABETES

## Its Causation and Treatment

(with special reference to India)

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BY

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TO THE MEDICAL AND NURSING STAFF OF A "CLASS IV LOCAL FUND DISPENSARY,"—HOWRAH GENERAL HOSPITAL.

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# PREFACE.

**Diabetes** is a disease of the greatest interest to any one working in the East, and it is surprising that there is not more literature bearing on the causation of this disease in India.

Until recently, that is to say, until Allen's researches were published, the treatment of glycosuria in India was most unsatisfactory. Some cases got better, apparently spontaneously ; others did not, in spite of treatment. This is not surprising in view of the extraordinary preponderance of carbohydrates in the average Indian diets.

Consequently, to ask an Indian vegetarian patient to live on a carbohydrate-free diet was to advise an impossibility. But, with the new conception of diabetes advanced by Allen, the whole position has changed, and there seems no reason why any intelligent Indian should not become, and remain, sugar-free.

An attempt has been made in the following pages to consider the disease from an Indian standpoint, and from the position of a practitioner in the East. Consequently, very little stress has been laid on elaborate chemical or physiological investigations, which, important and interesting as they may be, are entirely beyond the scope of the vast bulk of men practising in the tropics.

I wish it had been practicable to continue and extend the observations on blood sugar, but the exigencies of military duty removed my resident staff at a critical period, and made further observations impossible for the moment. The same remark applies to various chemicals required in testing—they are either unobtainable, or prohibitive in price.

In spite of these drawbacks, it is, I think, quite plain that the successful treatment of diabetes can be inexpensively carried out in an ordinary district hospital, and the patients educated to keep themselves sugar-free.

Lastly, my sincere acknowledgments are due to the distinguished physiologists and clinicians from whose writings I have quoted. I have drawn largely on the work of Dr. J. J. R. Macleod and Major McCay, I.M.S., for physiological facts, on Dr. Barker (*Monographic Medicine*), Dr. Cammidge (*Practitioner* and *Lancet*), Dr. Leyton (*Practitioner and Clinical Journal*), and on numerous valuable articles in the *American Journal of Medical Sciences* of 1915—1917.

I am much indebted to Drs. Mitra, Burdhan, and Bagchi for assistance in the clinical work and Indian diets recorded in this volume.

E. E. WATERS.

HOWRAH GENERAL HOSPITAL,
*November, 1917.*

# DIABETES.

## CHAPTER I.

Diabetes is a disorder of metabolism characterised by an inability to burn up sugar in the body, by an increase of sugar in the blood, and generally by the presence of demonstrable sugar in the urine. This, perhaps, is the only scientific way of defining the condition. It is better to regard it as a disease of metabolism, of which the glycosuria is an outward, and even a late sign, comparable to the albuminuria of kidney disease, to the ascites of cardiac failure, than to look on the glycosuria as the disease itself. For we know that many conditions may produce glycosuria—some natural, some artificial. But, from the sanction of long usage, diabetes has come to mean the presence of sugar in the urine, with the attendant symptoms and complications.

It has been known to writers for many years, particularly to early Hindu writers. In the Sushruta Samhita (which is variously dated at 400 B. C. and at 250 A. D.), diabetes is referred to as Madhumeha, and later in the same volume, directions for treatment are given.

We are told that Aretæus (A. D. 150) used the term diabetes (a syphon), and that Galen was familiar with the disease. In comparatively modern times the first rational description appears to have been that of Thomas Willis, who, in 1684, wrote of the "urine being exceedingly sweet, as if there had been honey and sugar in it." Dobson, a century later, evaporated urine and demonstrated actual sugar. Then, in 1849, Claude Bernard demonstrated that *piqûre* in the floor of the fourth ventricle caused a transitory glycosuria. In 1857, he discovered the glycogenic function of the liver. A few years later, in 1889, it was demonstrated that complete extirpation of the pancreas caused a persistent diabetes, whilst, in 1900, Opie asserted the connection between a degeneration of the islands of Langerhans in the pancreas and the occurrence of glycosuria.

Much more interesting than these historical statistics are the etiological factors in the disease, the questions of race and sex incidence. Diabetes is not a common disease in Europe: the mean annual mortality is not more than 5 per 100,000 living. But even in Europe there are wide variations. In Paris the rate is 14 per 100,000, in Copenhagen 7, in London 7, in Vienna 4, and in Naples 3. For England the rate is 7, Ireland 3, Scotland 2, Prussia 2, and Italy 1·5.

According to the text-books, diabetes is unknown amongst the Chinese and Japanese, and rare amongst African Negroes. Probably this means that the disease has not been looked for and statistics published. So far as China is concerned, it is reasonably common. Reid (A. J. M. S., April 1916) collected notes of 207 cases of diabetes from various missionary doctors. He thinks that the disease is less common than in the West, but that it is becoming more frequently recognized as European methods spread and as they are more widely adopted by the educated and wealthier classes.

In Korea, diabetes is known as the "Sweet-water disease."

Two classes of the community are notoriously prone to the disease, namely, the prosperous and educated classes of India and Ceylon, and the Jews. But with the Jews the liability is an acquired one—a disease of environment or occupation, not of race. There are, in and around Calcutta, large numbers of Jews, both fair and dark. These people have lived in India for generations, speak little or no English, and are not wealthy nor educated as a class. So far as I can find out, they do not suffer from diabetes, nor do I remember ever to have seen a case in a Jew. The Ezra Hospital, Calcutta, is reserved for Jews; yet, so far as my information goes, a case of diabetes is almost unknown.

The actual statistics for ten years are as follows :—

TABLE I.

*Ezra Hospital, Calcutta.*

| Year. | Total medical cases. | Cases of diabetes. |
|---|---|---|
| 1907 ... | 244 | 1 |
| 1908 ... | 176 | 1 |
| 1909 ... | 214 | nil. |
| 1910 ... | 103 | nil. |
| 1911 ... | 175 | nil. |
| 1912 ... | 196 | 1 |
| 1913 ... | 100 | nil. |
| 1914 ... | 126 | 1 |
| 1915 ... | 110 | nil. |
| 1916 ... | 104 | nil. |
| TOTAL ... | 1,548 | 4 |

It is plain from these statistics that Jews living in Bengal are not unduly subject to diabetes. If the disease does appear, it is due to other causes than race and climate.

Statistical evidence of disease in India and the East is notoriously unreliable and difficult to obtain owing to imperfect registration, to the lack of qualified doctors, and to the high proportion of deaths that occur without the attendance of any medical man.

Yet there are three classes of the community for whom statistics are available, and available in sufficient numbers to give us some information. These classes are the British Army in India, the Indian Army, and the Jail population.

These three groups are the more valuable in that they all have a liberal staff of trained medical officers eager to recognise and deal with any disability. Unfortunately, they do not contain the class which supplies the diabetic—the highly educated, somewhat indolent brain worker, the Indian lawyers, doctors, public officials, and land-owners.

The figures I refer to are to be found in Table XXXIII of the Annual Reports of the Sanitary Commissioner with the Government of India.

In 1912, the sick returns of cases of diabetes were these:—

| | | |
|---|---|---|
| British Officers with British Troops—Strength, 2,278. | Admissions | 1 |
| | Invalided | 0 |
| | Deaths | 0 |
| British Troops—Strength, 71,001 | Admissions | 7 |
| | Invalided | 3 |
| | Deaths | 0 |
| Wives of British Troops—Strength, 4,147. | Admissions | 0 |
| | Invalided | 0 |
| | Deaths | 0 |
| Children of British Troops—Strength, 7,046. | Admissions | 0 |
| | Invalided | 0 |
| | Deaths | 0 |
| British Officers with Indian Troops—Strength, 1,868. | Admissions | 0 |
| | Invalided | 0 |
| | Deaths | 0 |
| Indian Army—Strength, 148,200 | Admissions | 7 |
| | Invalided | |
| | Deaths | |
| Jail Population—Strength, 103,906 | Admissions | 3 |
| | Invalided | 0 |
| | Deaths | 0 |

These figures are very striking. There are over 86,340 Europeans of both sexes, and mostly under 35 years of age.

They have between them eight admissions for diabetes, three invalidings, and no deaths, so that, obviously, climate alone does not cause diabetes, nor does it favour the occurrence of the disease in young, presumably healthy, Europeans living on a mixed diet in the tropics.

### Indian Army.

The Indian Army numbers about a hundred and fifty-thousand men. It is composed almost entirely of young adults; perhaps a half of its strength are absolute vegetarians and teetotalers, whilst a large proportion of the other half touch meat at infrequent intervals. The men are scattered all over India and the East, yet have only 7 admissions for diabetes, with no invalidings and no deaths.

## JAILS.

The Jail population is generally one hundred to a hundred and ten thousand. It comprises all races and all classes. The lives are in no way selected, and many of them are broken down by disease, bad habits, or exposure. Yet there were only seven admissions and no deaths in 1912.

It should be remembered, too, that all these populations are floating ones. Drafts for the British Army come and go, recruiting and invaliding go on steadily in the Indian Army, whilst the Jail population is constantly changing. Thus, the actual number of individuals passed in review is much greater than the average strength. Again, each admission is shown in the returns, so that if one man in the Army reported sick three times during the year as the result of diabetes, he would register three admissions.

It is plain, then, from these statistics, and from those in the accompanying table, that climate alone is not responsible for diabetes, or that a purely vegetable diet will not cause it. We must search for some other factor.

### TABLE II.

*Incidence of Diabetes among British and Indian Troops and Prisoners.*

| | 1912. | | | 1913. | | | 1914. | | |
|---|---|---|---|---|---|---|---|---|---|
| | Strength. | Admissions. | Deaths. | Strength. | Admissions. | Deaths. | Strength. | Admissions. | Deaths. |
| British Officers with British Indian Troops. | 4,146 | 1 | ... | 4,212 | 3 | ... | 3,878 | ... | ... |
| British Troops... | 70,755 | 8 | 1 | 70,001 | 7 | ... | 60,581 | 3 | ... |
| Wives of British Troops. | 4,147 | 1 | ... | 4,213 | ... | ... | 3,772 | 1 | 1 |
| Children of British Troops. | 7,046 | ... | ... | 7,006 | ... | ... | 6,465 | ... | ... |
| Indian Army ... | 148,200 | 7 | ... | 158,526 | 12 | 1 | 168,767 | 5 | ... |
| Jail Population... | 103,906 | 3 | ... | 108,286 | 10 | 1 | 114,113 | 8 | 2 |
| TOTAL ... | 338,200 | 20 | 1 | 352,244 | 32 | 2 | 357,576 | 17 | 3 |

These figures are very striking. On an average population just over a third of a million and representing a very much larger number of individuals, the deaths from diabetes were two per annum and the admissions under twenty-three!

There are some factors which affect the incidence of diabetes:—

*Age.*—Glycosuria may occur at any age, but is most common in the fifth and sixth decades. The disease develops most frequently in early middle life, and the largest number of deaths occur from fifty to sixty-five.

*Sex.*—The disease is decidedly more common amongst males than females, especially in the middle years of life. In childhood and old age the numbers are much closer. American statistics for 1900 give 4,672 deaths from diabetes, of which 2,650 were in males and 2,022 in females. The English figures give 1,142 males to 869 females, with the same divergence in the middle years of life.

In the East, the disease is much less common in women than in men, or it certainly comes much less frequently to notice, which is not always the same thing. I have never seen a female case in hospital practice and can only remember one in private. Many Indian medical men, both of the allopathic and ayurvedic schools, tell me they have never seen a case of diabetes in a woman. There must be a reason for this, and the reason is probably to be found in the habits of the people. Diabetes, in Bengal at any rate, and the same is presumably true of other parts of the country, is a disease of the Bhadralog—the educated or gentlemanly class. This includes the land-owners, the lawyers, doctors, Government servants of the upper classes, businessmen, clerks, and so on. It occurs in the labouring classes, but only to a very limited extent, and is then often of the severe intractable type.

So notorious is this class distribution and so well-known the disease, that popular belief has it that "the Munsiff gets diabetes, the Deputy Magistrate doesn't," *i.e.*, the relatively active Deputy Magistrate, who frequently tours the country, escapes the disease, whilst the sedentary Munsiff or Sub-Judge, who sits in Court all day and writes his judgments in the evening, is particularly liable to it! It is not solely a question of excessive carbohydrate diet, for women are much stricter with their diet than men are, practically never take meat, only take fish as a relish, and proportionately eat more sweetmeats. Yet women rarely suffer from diabetes, Hindu widows never. Speaking generally, it is a question of over-eating, dyspepsia, want of exercise, and excessive mental work. Large numbers of the Bhadralog class are chronic dyspeptics, so that their food assimilation is perverted at the very beginning; they take little or no exercise, and they undergo long and continuous mental strain at their professions or businesses. Their women-folk, on the other hand, have ample time for their meals, and in one way or another take a considerable amount of exercise. Every Hindu wife, whatever her rank may be, takes an active share in the preparation of the family food, in the feeding and management of her children, and in similar household duties. Further, and this is an important point, every orthodox Hindu woman fasts once a month, with probably a partial fast on another day.

NOTE.—*Since writing the above, I am told that occasional cases of diabetes do occur in Hindu woman, even in Hindu widows. But my informants assure me that, when the disease does occur in women (and especially in widows), it is amongst those who are well to do. who* *live well, and who are burdened with the management of ownership of large estates. In other words, when the mental and physical conditions of an Indian woman assimilate to that of her male relations, then she is liable to diabetes.*

The austerities of the life of a Hindu widow, the frequent fast days and the scanty meals, are well known—and widows do not suffer from diabetes. Lastly, women, in India at any rate, are spared the brain work, the hours of continuous study, and mental exertion, of their male relations, which lay the foundations of the dyspepsias from which their men-folk so constantly suffer.

*Heredity* is generally supposed to be a factor of some moment in diabetics, and one frequently hears of the disease being present in several members of the same family. Fletcher quotes a series of cases in which hereditary influences were marked in 23·8 per cent.

This question is fully treated by Williams. (A. J. M. S., September 1917). He agrees with Jostin that the more carefully the family history is enquired into, the more commonly heredity is present. This, I think, agrees with our Indian experience.

*Infection* has often been cited as a factor in diabetes, but though cases do occur in husband and wife, there does not appear any reason for suspecting an infective element.

*Gout* certainly favours diabetes—or perhaps it should be said that the metabolic disturbances which occur in out tend also to occur in diabetes.

Attacks of glycosuria may alternate with gout, the patient may suffer from gout first and afterwards from glycosuria, or he may have gouty symptoms and glycosuria at the same time.

*Obesity* is a somewhat important factor, particularly in Bengal. Excessive carbohydrate diet, sugars, limited exercise, all favour stoutness, particularly in middle life. At this period exercise tends to be diminished, and the habits to become more luxurious, whilst business strain is often at its highest. In these circumstances glycosuria often appears, but it is not of a severe type and yields readily to treatment. On the other hand, obesity occurring in young persons may be accompanied by diabetes, and this is generally a serious and rapidly fatal complication.

*Syphilis* is said to favour diabetes, but I have met no definite case, and authorities in Europe differ as to the importance of this disease. The experiences discussed on page 31 are of much interest. Many other diseases have been followed by diabetes, particularly the infectious diseases. Enteric fever, scarlatina, cholera, diphtheria, are all noticed as having some relationship; malaria does not seem to be a common precursor, but influenza is said to precede many cases.

*Pregnancy* is occasionally complicated by glycosuria, though I have seen it only once. In *Exophthalmic Goitre* it is by no means uncommon, and indeed one would expect this to be the case, owing to the altered thyroid metabolism. Various drugs will cause a transient glycosuria; they will be discussed later.

The influence of the nervous system must be here referred to. The classical instance is that of Claude Bernard who, in 1849, performed the *piqûre* experiment.

This consists of puncturing the floor of the fourth ventricle between the centres for the vagus and auditory nerves. As a result there is hyperglycæmia, polyuria, and glycosuria, the latter varying in duration with the animal used, *i.e.*, it lasts some forty-eight hours in the dog, and six hours in the rabbit. It is thus not surprising that cerebral injuries are frequently followed by glycosuria, and many observers have noticed the relationship between head injuries and the disease. Organic lesions of the brain and cerebral hæmorrhage are frequently associated with glycosuria, even though the fourth ventricle be not affected. After an apoplexy sugar frequently appears in the urine, but generally clears up in a few days.

In a few instances tumours of the pons, cerebellum, and fourth ventricle have been recorded as causing diabetes, but these cases are medical curiosities rather than matters of daily interest.

# CHAPTER II.

## Physiology and Pathology.

At the commencement of the last chapter an attempt was made to define diabetes, and it was stated that one of the main factors in the disease was the inability of the organism to dispose of sugar. This is not yet generally accepted as an adequate definition, but the whole trend of modern investigation leads us to regard this as the essential. It has been realised for many years that other conditions besides true diabetes may cause the appearance of sugar in the urine, and it is from the study of some of these glycosurias that useful knowledge may be gained. Diabetes may be present and active for a considerable period before sugar appears in the urine. At first, the only disability is a low carbohydrate tolerance. The individual is unable to deal with a normal quantity of carbohydrate. Should the amount ingested be below the personal co-efficient, no outward sign of the disease is evident, but later, unless the carbohydrate intake is lessened, a chronic sugar poisoning goes on, and eventually even proteins are broken up and converted into sugar.

It is plain that many factors have to be considered in studying the probable causes of diabetes. We shall see that nervous influences may cause glycosuria, that it may follow disease of the pancreas, of the thyroids, and of other glands, that it may follow the injection of adrenalin and phloridzin, or the ingestion of an abnormal quantity of sugar.

First of all, let us discuss the sugar content of normal urine and then of normal blood. An ordinary specimen of urine, from the clinician's point of view, contains no albumin, no sugar, and a certain amount of urea, phosphates, chlorides, sulphates, etc. But, though there be no sugar when the ordinary tests are applied, yet, when more sensitive means are used, the presence of a reducing agent can generally be detected (opinions differ on this point).

Several tests for sugar have been introduced; they generally depend on the property possessed by glucose of reducing cupric to cuprous oxide in the presence of an alkali. The tests fall into two groups, the one represented by Fehling's method and including Pavy's, Trommer's, and Moore's tests, the second, and more delicate group, represented by the Benedict test and also including the Worm Müller, and the Nylander Hammarsten.

The use of yeast is not practicable in ordinary *clinical* work in the East, nor are the osazone and polariscope methods, though they are of course of the greatest value in laboratory work.

For routine clinical work, it is customary to depend on either the Fehling or the Benedict test for both qualitative and quantitative estimations. Fehling's test has the sanction of long usage and convenience, but it has many fallacies. It is not sufficiently delicate to demonstrate small quantities of sugar, and the results are to some extent vitiated by the presence in the urine of uric acid and urates, creatinin, urochrome, glycuronic acid, and the products of certain drugs of the coal tar series.

It is of the greatest importance to know whether, in doubtful cases, a state of glycosuria exists, for it is on the early detection of sugar, and consequent prompt treatment, that the patient's fate depends. For this reason Fehling's test is not sufficiently accurate, for it is reduced by the above-mentioned substances and does not give an accurate reading for small quantities of sugar—just those small quantities that it is most necessary to know of.

It is as yet unsettled whether there is any sugar in normal urine. According to some observers, normal urine contains ·05 per cent. of dextrose, but "as the matter stands at present we must conclude that there is no unassailable evidence that dextrose is a constituent of normal urine."—(Macleod).

Fehling's test will not detect a smaller quantity of dextrose than ·1 per cent., whilst Benedict's test will react to ·08 per cent. assuming that normal urine contains ·05 per cent. of dextrose.

According to Macleod, the addition of ·015 per cent. of dextrose to a sample of normal urine gives no reaction after two minutes' boiling with Benedict's solution, whilst ·03 per cent. gives a positive result.

It is proposed therefore only to describe two tests for dextrose, Fehling's and Benedict's, the former because of its world-wide use, the latter because of its delicacy and accuracy.

*Fehling's Test.*—The test fluid consists of an alkaline solution of potassio tartrate of copper, so prepared that 10 c.c. is reduced by 0·05 of glucose. It is generally put up in two solutions, No. 1 and No. 2—the alkali and the copper—which are mixed in equal parts at the time of making the test. To such a mixture add a few drops of urine and boil; continue adding urine until the Fehling and urine are in the proportion of two to one. The Fehling must always be in excess and the boiling not too prolonged. If, after such boiling, the solution remains clear, no *large* amount of sugar is present. The well-known yellow precipitate is due to cuprous hydrate ($Cu_2O, H_2O$) which on longer boiling becomes cuprous oxide ($Cu_2O$).

There are various fallacies to be guarded against, and they are of considerable importance.

(i) The urine must be free from albumin.

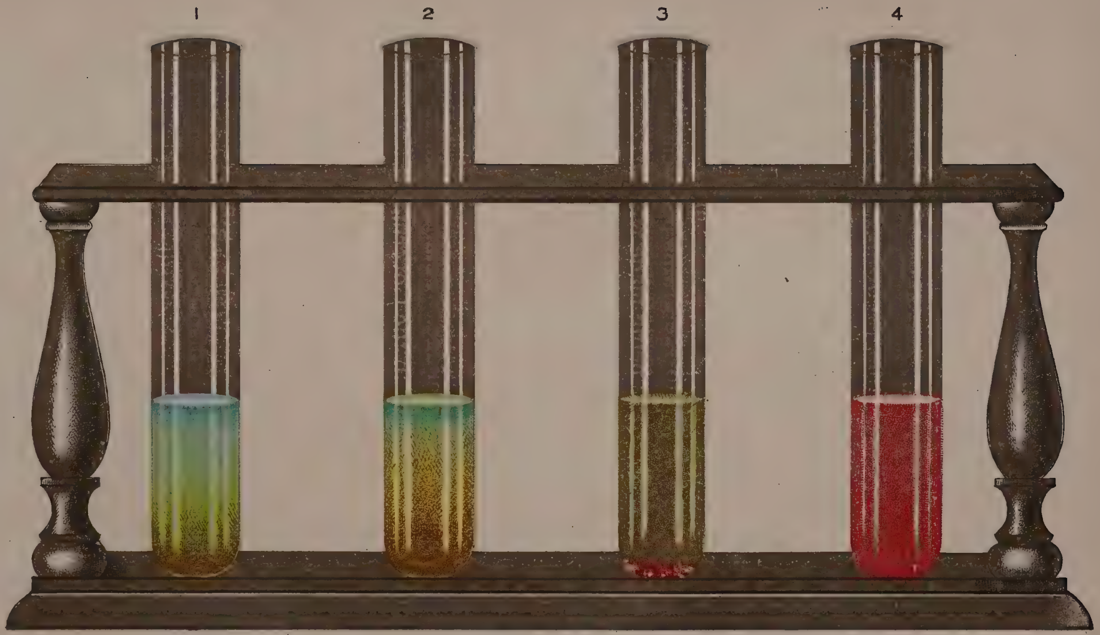
(ii) It must not be ammoniacal.

(iii) The presence of other reducing agents than sugar, *i.e.*, chloroform, chloral, and morphia may give a precipitate due to glycuronic acid. Uric acid and urates, creatinin, urochrome, and the products of various coal tar drugs, all act as reducing agents and must be allowed for.

These can be eliminated by the fermentation test, or by filtering the urine seven or eight times through a charcoal filter, by which means all reducing substances other than sugar are removed.

Fehling's solution is also in common use for the *quantitative estimation* of sugar. It is not absolutely accurate, but it is the ordinary every-day test in India. The method is as follows:—Take a sample of the 24-hour urine, dilute to 1 in 20 and fill a burette with it. In a white porcelain dish have 10 c.c. of Fehling's solution, diluted with water. Bring this to the boil, allow the urine to

Plate I.



Benedict's Test.

I. Sugar 0·5% or 2·2 Grains per ounce.
II. „ 2·0% or 8·8 Grains per ounce.
III. „ 4·0% or 17·6 Grains per ounce
IV. Diacetic Acid Gerbardt's Test.

run in drop by drop, stirring constantly, until the blue colour is discharged. Read off the amount of urine required and calculate. Thus, supposing 40 c.c. of diluted urine are required to decolorize 10 c.c. Fehling (equal to ·05 grm. glucose) then $\frac{40}{20} = 2$ c.c. urine contain ·05 gramme glucose. From this, knowing the total daily excretion of urine, it is very easy to calculate the total daily output of sugar. It is the total daily output that one wants to know in clinical work. A patient is not much interested in the difference between 5 per cent. and 2 per cent. of sugar, but if he be told that his sugar output has dropped from 1,200 grains to 150 grains per diem, he feels that some real progress is being made with his case, and is proportionately encouraged.

These are the qualitative and quantitative tests in daily use, but they are not sufficiently accurate for small quantities of sugar and other methods are required. Of these the most modern and most accurate is the Benedict test, as modified by Cammidge. Fehling's solution has been replaced by Benedict's in the Government Stores.

*Benedict's Test.*—The solutions required are these. Dissolve 200 grms. of sodium citrate, 100 grms. of anhydrous sodium carbonate, and 10 grms. of sodium bicarbonate in 600 c.c. distilled water with the aid of heat. Add to it, with constant stirring, a solution of 21 grms. crystallised $CuSo_4$ dissolved in 150 c.c. distilled water. When this mixture has cooled to room-temperature, it is made up to 1,000 c.c. with distilled water and filtered. If preserved in a tightly stoppered bottle, the solution will keep indefinitely and can be used for quantitative and qualitative work.

Other solutions are required. They are: (i) A saturated solution of sodium carbonate (to be added to the copper re-agent immediately before use). (ii) A twentieth normal solution of iodine. This must be carefully prepared. Dissolve 9 grms. of potassium iodide in 200 c.c. of distilled water, add 6·35 grms. of pure resublimed iodine, and shake until dissolved. Make up to 1,000 c.c. with distilled water. (iii) A corresponding twentieth normal solution of sodium thiosulphate is made by dissolving 13 grms. of the crystallised salt in 500 c.c. of distilled water and making up to 1,000 c.c. This solution is standardised against the iodine solution, preferably after standing over night, using a 1 per cent. starch solution as the indicator and diluting as necessary according to the formula $\frac{N \times D}{\mathit{N}} = C$, when N = quantity of thiosulphate solution left to be diluted (in c.c.), D = the difference between the actual and theoretical number of c.c. of thiosulphate required in the trial, $\mathit{N}$ = the number of c.c. actually used, and C = the amount of distilled water in c.c. to be added to bring the solution to the standard strength.

Very little apparatus is required for the estimation: a 200 c.c. conical flask is fitted with a rubber stopper perforated with two holes; through one of these passes a large headed thistle funnel, the other is left open to act as a vent. The stem of the funnel is drawn out to a jet and is so arranged that the tip of the jet is just covered when the flask contains 100 c.c. of water.

*Qualitative Test for Sugar.* (Benedict, modified by Cammidge.) Take 5 c.c. of Benedict solution in a clean test-tube and add eight drops, not more, of urine. Boil, either over a spirit lamp, or preferably in a water bath, for five minutes. By using a water bath several specimens can be heated at once.

After heating, allow the tube to cool at room-temperature for two minutes; if the urine is free from sugar the copper solution is clear, or only shows at the end of that time a faint *blue* turbidity due to precipitated urates.

If sugar be present the solution becomes more or less opaque and changes colour, either before or after cooling, the bulk, colour, and rapidity of formation of the precipitate varying with the percentage of sugar. If there be only a small amount, under 0·1 per cent., a light green opacity appears after the test-tube has been removed from the water bath; a more bulky green precipitate, present when the tube is taken out of the boiling water, indicates about 0·1 to 0·5 per cent.; a dense yellow precipitate shows about 0·5 to 2·0 per cent., and a thick red precipitate points to there being over 2·0 per cent. of sugar.

The *quantitative* estimation is a little more difficult. Having obtained a rough idea of the probable percentage of sugar the urine is diluted with water according to the following scheme:—

A. Light green pptte dilute ... 40 c.c. to 100 c.c.
B. Dense yellow ,, ,, ... 20 c.c. to 100 c.c.
C. Thick red ,, ,, ... 10 c.c. to 100 c.c.
D. Rarely ,, ,, ... 5 c.c. to 100 c.c.

Take exactly 5 c.c. of diluted urine and add it to 15 c.c. of modified Benedict solution, plus 5 c.c. of a saturated solution of sodium carbonate. Mix in a 200 c.c. beaker. Cover with a watch glass, place on wire gauze, and boil for three minutes over a Bunsen flame. After the exact period of boiling, run in cautiously 10 c.c. of pure HCl. (sp. gr. 1·16), taking care to avoid excessive frothing. The contents of the beaker are poured as rapidly as possible into the thistle funnel of the flask already described, which has been previously prepared by placing in it 100 c.c. of water and 20 c.c. of twentieth normal iodine solution, measured with a pipette kept for the purpose. Care must be taken that the copper solution does not run into the flask too quickly, and that it is thoroughly mixed with the iodine solution. The beaker is washed out with a few c.c. of hot water and the washings poured into the funnel.

The flask is now unstoppered, the stem of the funnel washed into the iodine with a little water, and the standardized 20th normal thiosulphate solution run into the latter from a burette until the brown colour of the iodine is almost discharged. At this point, five or six drops of fresh cold starch solution (about 1 per cent.) are introduced and the addition of the thiosulphate continued until the blue colour of the resulting cold starch is completely discharged.

When the end point is reached, the solution has a faint light green colour which is quite easy to distinguish from the blue of the iodo starch, even by artificial light. A control consisting of 100 c.c. and 20 c.c. of twentieth normal iodine solution, measured with the same pipette is titrated with the sodium thiosulphate solution in a similar way.

The difference between the number of cubic centimetre of thiosulphate used in the test and consumed by the control, multiplied by a factor varying with the dilution of the urine employed according to the following table, gives the percentage of sugar in the original urine.

If a 40 per cent. dilution (A) was used multiply by 0·0845.
20 ,, ,, (B) ,, ,, (2 × ·0845) = 0·169.
10 ,, ,, (C) ,, ,, (4 × ·0845) = 0·338.
5 ,, ,, (D) ,, ,, (8 × ·0845) = 0·676.

The total excretion for the day can then be worked out if the amount of urine passed is known.

This method, with a little practice, is fairly rapid, particularly when several specimens are being examined at the same time.

The solution is about ten times as sensitive as Fehling's solution, so that a small proportion of urine gives an unmistakable reaction, even when it contains a very low percentage of sugar.

It is not apparently reduced by uric acid, creatinin, etc., and is consequently much more accurate than Fehling's method.

Lastly, the same solutions can be used for the estimation of sugar in the blood and this estimation is being daily recognised as of even more importance than the amount of glycosuria.

*The Sugar in the Blood.*—It is now necessary to consider the question of sugar in the blood, and to recognise how important this is in estimating the probable causes of diabetes. That sugar exists in diabetic blood has been known for 140 years; whilst Claude Bernard in 1845 recognised it as a constituent of normal blood, and demonstrated that it was increased as the result of his *piqûre* experiment.

Recently, interest in this subject has revived, and some important results have been arrived at by Macleod and his assistants. It is clear that there is no direct relation between the sugar in the blood and that in the urine—provided the "threshold" is not overstepped, there may be a considerable hyperglycæmia without any glycosuria. There are many problems connected with the blood sugar that are physiological rather than clinical, such as the distribution of the reducing agent between the blood plasma and corpuscles, the chemical nature of the reducing substance, and so on. These all have a bearing on the causation of diabetes, and for a fuller account the reader is referred to Macleod's original work. Important work on this subject is also being carried out by McCay and his staff in the Physiological Laboratories of the Calcutta Medical College. The first report appears in the *Indian Journal of Medical Research* for July 1916.

Before describing the effects of various conditions upon the blood sugar, it is necessary to give a brief description of the method of estimating this substance. McCay (*loc. cit.*) uses the Shaffer modification of the Michaelis-Roux method, and claims very accurate results. I have adopted the Benedict-Cammidge method, largely on account of its simplicity and the possibility of using it as a *clinical* means of investigation.

*The Benedict-Cammidge method.*—The same reagents and apparatus are employed as for the determination of sugar in the urine, but the iodine and thiosulphate solutions are diluted with distilled water 1 to 50 (*i.e.*, to a thousandth normal) and it is an advantage to use a smaller beaker (50 c.c.) and a smaller flask (100 c. c.) and funnel for the iodine. As the dilute solutions do not keep well they should be prepared as required. For most cases of glycosuria and for normal persons 0·2 c.c. of blood is used, but when a very high blood sugar is expected only 0·1 c.c. should be taken. (These quantities may be obtained from the finger blood by using a hæmocytometer pipette.) In order to remove the albumin from the blood and secure a solution suitable for sugar estimation, it is laked by mixing it with a known volume of deci-normal acetic acid (1 c.c. of glacial acetic acid to 125 c.c. of distilled water), made up to a definite volume with a saturated solution of sodium chloride, boiled, cooled, and filtered. An aliquot part of the clear filtrate is employed for the sugar estimation. Exactly 3 c.c. of deci-normal acetic acid are placed in a clean test-tube and 0·2 (or 0·1) c.c. of blood is added, the pipette being washed out with the acetic acid two or three times by suction. The mixture is allowed to stand for at least five minutes and is subsequently made up to 10 c.c. by adding exactly 6·8 c.c. (or 6·9 c.c. if 0·1 c.c. of blood was used) of clear saturated sodium chloride solution. The test-tube is now placed in a bath of boiling water for a minute or two, then cooled in running water and its contents poured on to a small dry filter paper (5·5 c.m. in diameter). As the clot is apt to slide out of the tube suddenly and cause the solution to overflow the filter, it should be broken up previously by tapping the tube against the finger. The first few drops of the filtrate are generally turbid and should be returned, as an absolutely clear filtrate is required.

When a sufficient amount has come through, exactly 7·5 c.c. are measured with a pipette into a small beaker containing 1 c.c. of modified Benedict solution and 1 c.c. of saturated sodium carbonate solution. The beaker is covered with a watch glass, placed on a wire gauze over a Bunsen flame and its contents gently boiled for exactly three minutes. The flame is then removed and 2 c.c. of pure hydrochloric acid (S. G. 1·16) are cautiously added. The solution is *at once* poured into a thistle funnel flask containing 25 c.c. of distilled water and 20 c.c. of thousandth normal iodine solution, the same precautions as to mixing, etc., being taken as in the urinary sugar estimation. The beaker is washed out with 2 or 3 c.c. of hot distilled water and the washings poured into the funnel. The liquid adhering to the stem of the funnel having been washed into the flask with distilled water, the iodine is titrated with standardised thousandth normal sodium thiosulphate solution, using three drops of freshly prepared cold starch solution (about 1 per cent.) as the indicator when the brown colour of the iodine has been almost discharged. The end point is shown by the disappearance of the last faint trace of blue or purple, with a resulting colourless solution when the flask is examined against a dead white background by diffused daylight; artificial light does not give satisfactory results. In warm weather it is advisable to cool the flask in running water before titration as the last trace of blue is then more readily seen.

A control titration with 25 c.c. distilled water and 20 c.c. of thousandth normal iodine is carried out for each blood, or consecutive series of bloods, and the end points carefully watched. The difference between the number of cubic centimetres of thousandth normal thiosulphate used for the test and for the control, multiplied by 0·0203 (or 0·0406 if only 1 c.c. of blood was taken) gives the percentage of sugar in the blood.

With a little practice, this method is easily carried out, and the results appear to be consistent. The percentage of sugar blood revealed by this method is lower than that obtained by McCay. Provided that the observer adheres to the same method (which one is not a matter of very great importance), one can work out one's own standard and record variations from it.

Using Shaffer's method, McCay gives the following results:—

Average normal percentage of sugar in the blood of Bengalis is 0·130.

Taking 50 normal Bengalis, the minimum was ·087 and the maximum 0·18. Fifteen ranged between 0·11 and 0·12, whilst twenty-five were between 0·11 an 0·14 per cent.

According to Rona, Doblin, and Takahashi (quoted by McCay) the average is 0·08 per cent. with a maximum of 0·128 per cent. and a minimum of 0·048 per cent.

McCay further found that the blood sugar steadily increased with the body weight, *i.e.*, the fatter the Bengali the more sugar in his blood. He gives the standards of the average percentage of blood sugar, thus:—

| | | |
|---|---|---|
| Europeans ... ... | ... 0·080 | per cent. |
| Bengalis ... ... | ... 0·130 | ,, |
| Poor, working Bengalis | ... 0·120—0·125 | ,, |
| Fat, indolent Bengalis | ... 0·150 | ,, |

"In the potential glycosuric the administration of glucose by the mouth is almost invariably followed by a decided increase in the sugar content of the blood." (McCay.)

"In normal individuals, glucose given in large quantities, 2—3 grammes per kilo. of body weight, has little or no effect in raising the percentage of blood sugar, or in producing glycosuria to such an extent as to give a positive reaction with Fehling's test, so long as the individual is able to dispose of the extra quantity ingested."

McCay therefore concludes that the question of blood sugar is a personal one; in normal individuals the blood sugar may vary within fairly wide limits, say from 0·10 to 0·29, but in any specific case the safety limit of blood sugar may range only within very narrow boundaries. Any excess over the higher limit causes at once the appearance of sugar in the urine; this limitation depends on diets, personal considerations, hereditary tendencies etc.

There are thus two main contentions—

1. Frank's—that the sugar content of the blood must reach 0·20 per cent. before glycosuria occurs.

2. McCay's—that the blood sugar content is a personal factor and that in individual cases, or in persons of the glycosuric diatheses (if one may use such a term) a very much lower percentage (0·163 in 25 cases) will cause glycosuria.

*Conditions influencing the amount of Sugar in the Blood.*—Apart from the methods of estimating blood sugar, we must consider various conditions which affect the amount of blood sugar and in their turn have a bearing in glycosuria. Normally, sugar is stored in the liver and muscles as glycogen, which can be discharged with the blood as sugar whenever required. Should the amount of blood sugar exceed a certain (arbitrary) point, the so-called renal threshold is overpassed and sugar appears in the urine. The rate of urine formation affects this glycosuria; when this is slow, sugar may accumulate in the blood, but, once the glycosuria has commenced, it quickly increases out of all proportion to that in the blood—the sugar acting as a poison or irritant.

*The Nervous Control of Blood Sugar.*—In spite of the constantly varying amounts of sugar and carbohydrate taken as food, the amount of blood sugar in health remains practically constant. This implies a sugar-controlling mechanism regulating this function, by which the blood sugar remains at a constant level even as the body temperature remains the same under widely differing external conditions. There is a certain amount of experimental evidence that there is a direct nervous control of the blood sugar. The most famous experiment is the *piqûre* of Claude Bernard, which is always followed by hyperglycæmia and glycosuria.

According to Macleod the results of this *piqûre* may be:—

(*i*) Hyperglycogenolysis due to increased blood supply to the liver.

(*ii*) Asphyxia and consequent splitting up of glycogen.

(*iii*) Increase of adrenalin secretion with similar consequences.

There is no doubt that increased blood supply and asphyxia both cause a hyperglycæmia, but it is also certain that increased adrenal secretion does the same. From Stewart's experiments it is clear that stimulation of the great splanchnic nerve causes an increase of adrenalin in the suprarenal veins, and this in its turn causes hyperglycæmia. Adrenalin will cause hyperglycæmia even when all nervous paths from the medulla to the abdominal cavity are cut. But the question has also been discussed whether medullary stimulation causes hyperglycæmia not through a direct action on the liver, but indirectly by increasing the secretion of adrenalin. Stimulation of the hepatic nerves causes a striking hyperglycæmia within a few minutes, whether the fibres central to the point of stimulation are cut or uncut. On the other hand, such stimulation has no effect in the absence of the adrenal glands. In the entire absence of adrenalin from the blood it is impossible to excite hyperglycogenolysis by stimulation of the nerve supply to the liver, whether by *piqûre*, splanchnic stimulation, or hepatic stimulation. (The only exception to this is by the administration of diuretin.) It may be said that the adrenals secrete adrenalin into the blood and that this secretion is under nerve control.

Macleod has made elaborate experiments on this point and sums up his conclusions thus: "The sympathetic nerve terminations that control the glycogenic function can be stimulated either by an excess of adrenalin in the blood, or by nerve impulses, but the latter are incapable of acting unless there is a certain concentration of adrenalin in the blood."

*The Ductless Glands and Sugar Metabolism.*—We have seen that the nervous system, directly or indirectly, has a marked influence on the blood sugar. We must now consider whether the ductless glands affect metabolism in any way. It must be remembered that carbohydrates are digested and absorbed in and from the intestine, the sugar products are carried to the liver, stored there as glycogen, and finally discharged again as sugar into the blood stream. It is possible that as the blood sugar falls, the deficiency stimulates the liver to deliver more sugar into the blood—the dextrose acts as a hormone. But there are other secretions which admittedly influence the glycogenolytic process. These are chiefly the products of the adrenals, the islands of Langerhans, the parathyroids, and the internal secretions of the thyroid and hypophysis cerebri.

In addition to these, carbonic and lactic acids, and the inorganic salts may affect the secretion.

*The adrenal secretion.*—Reference has already been made to the relationship between the nervous system and the adrenals. But it has been shown (Blum, Herter, and Richards) that the intravenous or subcutaneous injection of adrenalin causes hyperglycæmia and glycosuria, though the appearance of urinary sugar diminishes after repeated injections. It seems that the effect of the adrenalin is due to a rapid conversion of the glycogen into blood sugar, this action taking place on both liver and muscle glycogen. Ringer, for example, found that when animals are made glycogen-free by fasting, exposure to cold, and repeated injections of phloridzin, no increase in glycosuria results from intraperitoneal injections of adrenalin; also adrenalin causes glycosuria when all nervous paths from the medulla to the abdominal cavity are severed.

Only contradictory results have been obtained regarding the amount of blood sugar in Addison's disease.

*The Influence of the Pancreas.*—The relationship of the pancreas to diabetes has long been studied, and pancreatic changes in severe cases of the disease have been frequently recorded. It is established that when the pancreas is *completely* removed in dogs, a severe intractable and rapidly fatal diabetes occurs. But if the processus uncinatus, which has no intimate connection with the duodenum, and no duct, is stitched into the abdominal wall, and the remainder of the pancreas removed, then no diabetes occurs. The experiments of Lombriso and Macallum proved that the nerve connection was not essential in preventing glycosuria, for an absolutely independent pancreas graft prevented glycosuria, whilst on its removal a rapidly fatal diabetes developed. What then is the action of the pancreas? Is it through an internal secretion, or is it a local action by it on substances carried in the blood? The general weight of evidence appears to be in favour of an internal secretion from the pancreas regulating the carbohydrate metabolism, and that possibly this hormone is absent frcm diabetic blood.

It was suggested by Opie in 1900 that in diabetes the disease was confined to the islands of Langerhans. These structures have special staining properties and a rich blood supply. They are placed towards the centre of the pancreatic lobules. They are believed by some authorities to be separate structures, whilst others

(Dale) assert that they are merely transitional cells, in process of development into ordinary secreting cells.

There are three main theories regarding the influence of the pancreas in diabetes :—

I. There may be a specific atrophy of the glandular parenchyma, combined with an inflammatory process in the gland. (Hansemann.)

II. The insular theory, which locates the main lesion in the islands of Langerhans. (Opie, Weichselbaum.) They are said to undergo both qualitative and quantitative changes.

III. The most modern theory is that all the epithelial tissues of the pancreas, secreting tubules, as well as islands are implicated in the changes which lead to diabetes. Allen supposes that the disease results from an exhaustion of the pancreatic cells. (Williams, A. J. M. S., September 1917.) This is quite consistent with the chronic dyspepsia of the educated Indian and his carbohydrate-laden diet.

It must not be forgotten that, as Macleod shrewdly remarks, the pancreatic changes may be the *result* of diabetes and not the cause, comparable to diabetic cataract and gangrene.

In this connection some recent work of Warthin and Wilson on the coincidence of syphilis and diabetes is worthy of attention (A. J. M. S., August 1916). They give detailed autopsies of the fatal cases of diabetes recently occurring in their practice. There were six cases ; all six presented histological changes of syphilis ; in four of them spirochætes were found in the myocardium, and in one of them in the pancreatic lesion. It is unnecessary to detail the cases here ; they were all severe cases, much more severe than the cases we ordinarily see in India. In all the pancreatic changes were marked. Briefly, they were :—

(1) Atrophy with fatty infiltration.

(2) Increase of stroma, both inter and intra lobular, of a patchy character.

(3) Inflammatory infiltrations, small in size, in the interlobular connective tissue and within the lobules.

(4) Changes in the islands of Langerhans were present in all six cases. The islands were either completely or very nearly atrophied. The destroyed islands were replaced by irregular masses of fibrous tissue showing no remains of the island structure.

(5) The acini showed marked changes in all six cases. Some were atrophic, but the majority were larger than normal.

(6) Numerous structures like newly formed islands were found ; they were new formed lobules of acini arising from the ducts. They represent attempts at regeneration.

(7) The blood vessels always showed varying degrees of sclerosis.

(8) Ducts were dilated and showed thickened walls.

Are the pancreatic changes the cause of the diabetes, and is syphilis a chief factor in the etiology of diabetes by causing the inter-acinar pancreatitis, which, according to Opie, is most frequently associated with the disease? To answer this question the authors set themselves to examine the pancreas in thirty-nine cases of old latent syphilis. A preliminary survey showed that the *pancreas was not normal in a single one of these old cases of syphilis.* The process is always a patchy one, but the tail and body of the organ are always more involved than the head.

The conclusion is that pancreatitis is associated with old latent syphilis and that the pancreatitis is local and patchy; that diabetes may be associated with the more marked degrees of syphilitic pancreatitis (and possibly of other internal organs). *That latent syphilis is the chief factor in the production of the form of pancreatitis most frequently associated with diabetes, but that diabetes is not always coincident with severe degrees of this type of pancreatitis.*

There are many conflicting facts to reconcile; in many typical cases of diabetes no lesion of the pancreatic cells can be found, and on the other hand, in complete destruction of the pancreas by necrosis or hæmorrhage, diabetes is frequently absent.

There is no doubt that the glycogenic function is upset by the removal of the pancreas, but this does not necessarily mean that interference with the glycogenic function is the only cause of diabetes, for, as we know, diabetes persists when carbohydrates are withheld, the sugar being derived from protein (Macleod). It may be that the process of glycolysis is first interfered with, the glycogenesis only secondarily.

*The action of other gland secretions.*—We have already discussed the importance of adrenalin in relation to the glycogenic function; has it any bearing on the pancreatic function. Here again, Macleod's work is of great value: "The pancreas and adrenal glands evidently influence sugar metabolisms in an opposite sense, for the former keeps down the sugar concentration in the blood, whilst the latter tends to make it rise." It may thuseb considered that the pancreas would, so far as its sugar relationship is concerned, neutralize the action of the adrenalin, so that after pancreatectomy, hyperglycæmia occurs simply because the internal secretion of the suprarenal glands acts unchecked on the sugar output of the liver. (Zuelzer.)

1. If the suprarenal glands are removed or the veins tied, removal of the pancreas causes little or no glycosuria in dogs.

2. Simultaneous injection of pancreatic extract and of such amounts of adrenalin as would alone lead to glycosuria is not followed by this condition.

These conclusions are generally modified by the intense shock caused by the removal of the pancreas.

Other investigators have confirmed some of Zuelzer's results, but find that though injection of finely disseminated pancreatic tissue renders adrenalin incapable of producing glycosuria, yet a similar inhibitory effect was produced when any irritating substance, such as hirudin, colloidal lymphogogues, or turpentine, was injected intraperitoneally. It is possible, therefore, that the irritation caused by the intraperitoneal injections of the extracts, etc., depressed the excretory function of the kidney towards sugar.

"To sum up, there is not the slightest *experimental evidence* that the hyperglycæmia, etc., following pancreatectomy is due solely to an uncontrolled action of the internal secretion of the adrenal gland."—(Macleod).

But, besides the suprarenals, the thyroid, parathyroid, and pituitary glands must be considered, as they all have some influence on the metabolism of carbohydrates.

It has long been known that when the thyroid and parathyroid glands are removed there is a marked lowering of tolerance to sugar. Later investigations made it seem probable that the parathyroids were responsible for this change, for when the thyroid and only two parathyroids were removed the carbohydrate tolerance was unaltered. But if three, or the whole four, parathyroids were removed, then the assimilation limits of carbohydrates were much lowered. It may be that the thyroid exercises a contrary action to the parathyroids, *i.e.*, that it *raises* the sugar tolerance, but there does not seem to be much evidence in favour of this view.

It is said that in Grave's disease the sugar tolerance is much lowered, whilst in myxœdema it is raised.

In all probability the parathyroid glands exercise a control over carbohydrate metabolism in such a way as to prevent the accumulation of excessive amount of dextrose in the organism, but that the thyroid glands, in so far as carbohydrate metabolism is concerned, have no direct influence whatever (Macleod p. 104).

*The Pituitary Gland.*—Acromegaly, which is a result of disease of the pituitary gland, is frequently, in the early stages, associated with glycosuria, but though this fact is established, it is extremely difficult to determine which part of the gland is at fault. If the posterior part of the gland be removed in a dog, polyuria and glycosuria occur, possibly from manipulation of this portion and consequent increased secretion. The secretion of the anterior part of the gland does not appear to have anything to do with carbohydrate metabolism. That is as far as the present state of our knowledge allows us to go.

*Renal Glycosurias.*—There are still other factors which affect the possible flow of sugar into the urine; as we have above discussed (p. 27) though there may be a considerable amount of sugar in the blood, very little gets through the renal cells into the urine (none at all according to some observers).

What is the reason for this? It is said that blood sugar is in a free chemical state, and that the kidney cells possess physical or chemical properties which render them impervious to such amounts as ordinarily occur in the blood. Possibly this is the case, for we have the analogy of the excretion of sodium chloride which appears to depend, not on the total amount in the blood, but on the degree to which this amount exceeds a certain limit.

With the fall of the blood salt content, the urinary excretion falls to the minimum.

But it has also been stated that glucose exists in the blood in a peculiar colloidal combination which cannot pass the renal epithelium—thus resembling albumen. There is no definite experimental evidence of this—it is a plausible theory, that is all. We have spoken above of the "renal threshold." This is an imaginary barrier which in normal circumstances prevents the excretion of the normal blood sugar into the urine. In the renal glycosurias, though the amount of blood sugar is no higher than is normal, yet the renal threshold is lowered and glycosuria occurs. This is obviously a different condition from that in which the blood sugar is much higher than normal and then the *excess* appears in the urine.

*Phloridzin Glycosuria.*—Phloridzin is a glucoside extracted from various rosaceous trees. It occurs in pinkish white crystals, slightly soluble in water, and has a dosage of 5 to 15 grains.

It was first shown by von Mering in 1886 that the administration of this glucoside to animals was followed by the appearance of dextrose in the urine. The amount of sugar excreted may be far in excess of that contained in the glucoside, and a large amount of the phloridzin may be recovered from the urine. From Frank's experiments it seems that phloridzin steadily reduces the amount of the blood sugar.

According to Mosberg, the drug acts on the renal tubules, for, if the glomerular vessels in frogs are tied, injections of phloridzin still produce glycosuria, whilst injections of glucose do not. Chemical analysis has demonstrated that phloridzin kidneys show a relatively large amount of sugar in the medulla, whilst if the tubules are blocked by certain dyes, such as carmine and methylene blue, the action of subsequent phloridzin injections may be negatived.

Now this action of phloridzin is remarkable, for the effect of a mere injection of the drug is to break down the barrier or threshold between the blood sugar and the urine and allow the sugar to be excreted—and this in a healthy man. It is difficult to decide whether this profound alteration is due to change in the blood sugar or in the renal epithelium, but the trend of modern opinion is to ascribe it to the renal tubules. The loss of blood sugar is followed by a diminution in both the hepatic and muscular glycogen, although the liver glycogen does not disappear altogether.

It was demonstrated by Lusk that, if sufficient doses of a phloridzin be given at suitable intervals, all the food sugar and all the sugar formed in the body may be eliminated by the kidneys. It seems as if the low sugar concentration were responsible for the inability to utilise dextrose, for, if excessive carbohydrates be given, considerable amounts may be utilized. If these excessive carbohydrates are not given, there is utilisations of body proteins and much increased excretion of nitrogen. Acidosis and coma may also occur.

The glycosuria thus produced by phloridzin is, when persistent, of a severe type, more severe even in its way than that produced by pancreatectomy. Other drugs such as corrosive sublimate, uranium salts, and the chromates cause a similar but milder glycosuria, without increasing the blood sugar.

A few cases of renal glycosuria have been described in man, but later investigation has thrown considerable doubt on them. It is possible that the glycosuria of pregnancy, which is notoriously mild and not uncommon, is of the nature of a renal glycosuria, for there is no increase in the blood sugar.

The glycosuria of adolescents, which we see occasionally in examining candidates for the public services may be of this nature.

The following is Rosenberger's classification of substances that will produce glycosuria. The majority have been observed only in animals, but a few are known from observation in man :—

I. Kidney poisons : phloridzin, uranium salts, chromium salts, cantharides.

II. Asphyxial agents : curare, methyldelphinin, acetone, hydrocyanic acid, carbon monoxide.

III. Nerve poisons: salts, diuretics, hypnotics, and narcotics (chloral, ether, etc.), convulsive agents.

IV. Liver poisons: phosphorus, alcohol.

V. Unknown mode of action: atropin, amyl nitrite, acids, etc.

*Alimentary Tolerance and Alimentary Glycosuria.*—The ordinary storage capacity (for glycogen) (of the human liver) is 150 to 200 grammes, so that it is normally able to deal with any average amount of sugar coming from the intestine. This power is apt to be upset if the glucose is absorbed too rapidly or the personal glucose coefficient is too low.

Whilst in the normal individual the glucose in the portal vessels is absorbed and dealt with sufficiently rapidly to prevent a hyperglycæmia and consequent glycosuria, in some persons this process is interfered with so that the amount of blood sugar rapidly increases and a glycosuria occurs. This condition is known as alimentary glycosuria.

Associated with this condition is that of *sugar tolerance.* Most of us take an excessive amount of carbohydrate and sugar, but, unless the personal limit or coefficient is exceeded, this is burnt up in the body and no glycosuria appears. Normally, it is said, about 100 grammes of sugar may be taken in one dose without causing glycosuria, but I have frequently given much larger amounts (up to 300 grammes of sweetmeats) to Indian students without producing any urinary sugar. Apparently, the tolerance is lower in young children and elderly people than it is in adolescents.

Various conditions affect the capacity for dealing with carbohydrates and sugar taken as food. It is not uncommon to find a moderate amount of sugar in young candidates for the public services; on enquiry it may be found that the candidate has taken a meal of sweetmeats whilst waiting for his interview, and that this (ordinary) amount, plus the nervous influence of the impending medical examination has been sufficient to cause a temporary lowering of the toleration limit and a glycosuria. Alcoholism may lower the alimentary tolerance, particularly alcohol in the form of beer. Accidents are frequently followed by a temporary glycosuria, and so too are acute infections, especially pneumonia. On the other hand, grave disease of the liver, such as liver abscess, cirrhosis, etc., does not cause any alteration in the sugar tolerance.

There are three main factors which regulate the significance of this toleration limit; they are:—

1. The rapidity of absorption from the alimentary canal.

2. The ability in the part of the body to remove an excess of glucose from the circulating blood.

3. The threshold limit at which the kidneys begin to excrete the excess into the urine.

*The Influence of Lævulose and Galactose.*—Both of these sugars have a toleration limit in man similar to that of glucose and similar experiments can be performed with them. Lævulose is converted by the liver into glycogen and stored there; if lævulose be given to fasting animals after the pancreas has been removed, there is an increase of hepatic glycogen. The liver is especially concerned in lævulose assimilation, more so than is the case with glucose (de Filippi). The toleration limit for lævulose in man is about 120—150 grammes; when 100 grammes are administered to normal men, some 15 per cent. discharge lævulose in the urine.

Lævulose has been used as a test of the hepatic function; whereas in normal individuals only 15 per cent. show lævulosuria after a dose of 100 grammes, in hepatic cirrhosis the percentage is 86, in catarrhal jaundice, and chololitheasis 91, in syphilitic liver 61, whilst in passive congestion of the liver it is down to normal at 16 per cent.

*Galactose* has also been used as a test of the hepatic function. The toleration limit for man is lower than in the case of lævulose, being only 20 grammes or thereabouts. When the amount administered is in excess of this normal limit, considerable quantities pass into the urine. The indications are very similar to those of lævulose. In catarrhal jaundice galactosuria appears in 99 per cent., in hepatic cirrhosis in 70 per cent., in gallstones, 17 per cent., in all other liver conditions galactose hardly appears in the urine at all after the administration of normal doses.

## *Proteins and Metabolism.*

Whilst normally the body forms sugar from carbohydrates, yet it has the power, under certain conditions, of forming sugar from proteids (and, according to some, from fats). It has been repeatedly shown that in animals from whom the pancreas has been removed, or to whom phloridzin has been steadily given, that the amount of sugar excreted during carbohydrate starvation has been far in excess of any that could have been stored in the tissue as glycogen.

Whence does this sugar come? It is maintained by Van Noorden that most proteids contain a carbohydrate group and that this group is split off during digestion; the cleavage occurs during the action of the pepsin-hydrochloric acid, for albumose does not contain carbohydrates. The carbohydrate thus split off behaves just like a normal carbohydrate; it is absorbed and goes to the liver. Certain albumins which are particularly rich in carbohydrate groups, *e.g.*, egg-albumens, do not especially affect the sugar formation; the influence is relatively large when the albumen is taken raw, but very slight when it is coagulated by heat. On the other hand, some proteins act as marked excitants of sugar production, for example muscle albumen, which contains very little carbohydrate, and casein, which hardly contains any. This can be demonstrated by administering them to depancreatised animals or to those treated with phloridzin.

This theory of Van Noorden's is flatly contradicted by Hewlett, who says it has been definitely abandoned, because the amount of sugar eliminated bears no constant relation to such preformed carbohydrates. It appears that the protein molecule is made up of a large number of simpler compounds, chiefly amino-acids. During the process of protein disintegration in the body, these simpler compounds are liberated, and it is from these units that the body forms glucose by a process of deamination and synthesis. It has been shown by Ringer and Lusk that certain of these units, such as glycocoll alanin, aspartic and glutamic acids are converted more or less completely into sugar in phloridzinised dogs.

*The D : N ratio.*—By this expression is meant the relationship between the urinary dextrose and the urinary nitrogen, and it is used to determine the amount of sugar which is derived from proteids in the body.

To discuss the process of glyconeogenesis, it is necessary to have an animal that is completely diabetic, either from true diabetes, from phloridzin, or by depancreatisation. Generally, an animal is regarded as completely diabetic when the ratio of dextrose to nitrogen is 2·8 to 1 or more. Were all the carbon present in the protein molecule converted into dextrose, relatively large amounts of glucose could be formed from proteids. If all this sugar appeared in the urine the D : N ratio would be nearly 8 : 1; this is impossible, but more than 45 per cent. of the carbon as represented in the ratio of 2·8 to 1 can be excreted this way, for in phloridzin diabetes, ratios of 3·2 : 1 are not infrequent, showing that 60 per cent. of the protein carbon is appearing as sugar. Incarrying out these investigations careful estimations are made of the urinary nitrogen and dextrose; the animal is then deprived of glycogen by starvation, phloridzin, etc. It is then fed with the substance to be investigated; if this substance contains no nitrogen and causes no change in the nitrogen excretion any increase in that of the dextrose must represent the extent to which it has been converted into sugar. On the other hand, if the substance contains nitrogen, some of this is derived from the body protein, and some from the administered substance.

In human diabetics of a severe type the D : N ratio shows considerable variation both in different patients and from day to day. This is possibly due to the varying amount of sugar or protein retained in the body. In a large number of human cases the D : N ratio is less than 3·85 : 1, but recent investigations by Falta and Grafe have shown that D : N ratios of 5 and over may persist for some time. It is not easy to give an explanation of these high ratios, for the errors in the human subject must be many, but either an unusual quantity of sugar must be formed from proteins or fats must be converted into glucose (*A. J. M. S.* January, 1917).

*Glyconeogenesis from fats.*—As to whether this occurs, considerable uncertainty exists. Fats are made up of glycerin combined with fatty acids. It is undoubted that the glycerin may give rise to sugar, but there is no evidence that sugar can be derived from fatty acids. The feeding of glycerine to depancreatized dogs causes an increase in the output of sugar (Nuthje). The question is discussed by Van Noorden, whose main conclusion is that the liver uses fats for the purpose of forming sugar only when the poverty of other materials makes it necessary. Allen (*A. J. M. S.* March, 1917) also gives a complete exposition of the most modern views on this important point.

*The Respiratory Quotient.*—In discussing the question of food-stuffs, the relation between the amount of oxygen consumed and the quantity of carbon dioxide eliminated is found to vary with the kind of food ingested. This ratio, which is known as the respiratory quotient, is for carbohydrates 1·0, for proteids 0·8, and for fats 0·7.

Now the energy set free in the body may be estimated if we know the quantity of carbohydrate, of fat, and of protein consumed in a given time, for each gramme in burning sets free a certain amount of heat. If the ordinary food-stuffs are oxidised to the same extent as in the body we get the following results (Hewlett):—

| | | | | | |
|---|---|---|---|---|---|
| One | gramme | glucose | yields | 3·8 | calories. |
| ,, | ,, | cane sugar | ,, | 4·0 | ,, |
| ,, | ,, | starch | ,, | 4·1 | ,, |
| ,, | ,, | fat | ,, | 9·3 | ,, |
| ,, | ,, | protein | ,, | 4·1 | ,, |

In order to determine the total metabolism it is necessary to estimate the amount and kind of material used in the body. The nitrogenous waste products in the urine shows the rate of protein metabolism; the absorption of oxygen and elimination of $CO_2$ through the lungs show the amount of carbohydrates, fats, and the non-nitrogenous part of the proteins, for these are all burned to carbon dioxide and water, and the former leaves the body in the expired air. The separate amounts of fats and carbohydrates burned are estimated by determining the ratio between the volume of $CO_2$ given off from, and the volume of oxygen absorbed by, the lungs. This ratio is called the respiratory quotient (Hewlett):—

$$\text{Respiratory Quotient} \quad \ldots \quad \frac{\text{Vol. CO}}{\text{Vol. O}_2}$$

A high respiratory quotient indicates a predominance of carbohydrate metabolism, while a ratio between 0·7 and 0·8 indicates the predominance of a fat-protein combustion. In complete starvation, after the main portion of the glycogen reservoirs have been exhausted, the ratio falls to 0·74 or less, owing to the combustion of the fats and proteins of the body.

In diabetes the respiratory quotient tends to be reduced, and in a series of nineteen severe cases quoted by Benedict, the quotient was 0·74, as opposed to 0·84 of normal individuals. In normal men and animals the consumption of a large carbohydrate meal causes the respiratory ratio to rise rapidly, so that it approaches or equals 1·0.

In severe cases of diabetes, on the other hand, no such marked rise occurs after the administration of carbohydrates, and in the most severe cases the respiratory quotient is almost unaffected by the ingestion of such food-stuffs.

In severe diabetes then, the respiratory quotient is such as one finds when the patient is living on a protein-fat diet.

In other words, the patient has lost the power of dealing with carbohydrates and is using his proteins and (fats) in their place.

On the other hand, it may be due to inability of the liver to transfer oxygen to the fat and protein molecules. The importance of these theories will be evident later when treatment is discussed. There is no doubt that proteids (and possibly fats) supply the fuel upon which the diabetic lives, and it is probable that proteins are more available than fats. During the development of the diabetic state, as the animal is losing the ability to burn carbohydrates, it first of all falls back on proteins, and when this no longer suffices to produce the necessary calories, fat may also become involved.—(Macleod.)

# CHAPTER III.

## THE CLASSIFICATION, SYMBTOMS, AND COMPLICATIONS OF DIABETES.

Diabetes is one of the most difficult of diseases to classify—to label properly and put into its proper pigeon hole.

We have seen that the condition may vary from a mere transient glycosuria to the severest metabolic disturbance in which no carbohydrate can be utilised.

Taking a general view of the physiological principles enunciated in the last chapter, we may say that the rate of discharge of glycogen from the liver into the blood stream is controlled by many factors, some of which stimulate, whilst others depress the excretion. These factors are the pancreas, the chromaffin system (adrenals), the thyroids, parathyroids, the hypophysis cerebri, and possibly some cerebral centres. In normal conditions these agencies are fairly balanced, so that the discharged sugar into the blood stream amounts to 0·06 to 0·125 parts per cent.

Of these organs the pancreas tends to diminish the sugar consumption, the adrenals tend to increase it. But the pancreas is controlled by the thyroid, the parathyroids, and the hypophysis, whilst the adrenals are under the control of the central nervous system. All these controls, except that of the central nervous system, are exercised through the secretions of the various glands (hormones) conveyed through the blood stream.

It is plain that a temporary derangement of any one of these glands may upset the balance and result in a glycosuria, which may develop into a diabetes.

We may attempt a pathological classification of diabetes by recognising two main classes—one in which the blood sugar is much increased, causing a hyperglycæmia, and then a glycosuria, and the second, in which the blood sugar is normal or less than normal, but the kidneys fail to retain the sugar in the blood owing to the lowness of the renal threshold. Again, we may accept a clinical classification, and in this there are three principal classes:—

*Firstly*—Those who do not pass sugar in ordinary circumstances, but who can be made to excrete sugar by a dose of glucose which has no effect on ordinary individuals.

*Secondly*—Those in whom glycosuria occurs from time to time independently of the ingestion of excessive sugar.

*Thirdly*—Those in whom the glycosuria is continuous, or is suspended only temporarily by the observance of a more or less rigid dietary.

There is no sharp dividing line between these various classes, and one tends to pass insensibly into the other. No dose of starch which can be taken causes glycosuria in a normal person, it is not converted sufficiently rapidly to upset the normal tolerance; when starchy food causes glycosuria there is a definite morbid process present. On the other hand, if a patient shows lowered glucose tolerance in the course of an acute disease, it does not necessarily imply that he will develop diabetes later on.

In the course of an acute disease, any one unit in the pancreas-liver—adrenal chain may be disorganized, and a temporary glycosuria occur.

From the *clinical* point of view, therefore, diabetes is the presence of sugar in the urine; the case may be slight or severe, but the outstanding factor is the sugar, and this requires definite attention. From what symptoms is sugar to be suspected; what is it that causes the patient to consult a medical man? There may be no symptoms whatever, and the sugar may be detected only when a routine examination is made of the urine. Then, after further enquiry, the patient may admit that he has to get up at night to pass water, or that he has an abnormal appetite.

In India an extremely common early symptom is a burning sensation in the hands and feet, with some heaviness in the legs. There is decided inability for prolonged mental effort, and physical exertion is reduced to a minimum. With Europeans, often the first thing to attract attention is the collection of ants about the sugar-laden urine; this is by no means an infallible sign, though it has frightened many a man into having a complete analysis made of his urine.

Rapidly failing sight may attract attention, vague "rheumatic" pains may be noticed, and sometimes a coldness of the legs and feet. The raw tongue, scanty saliva, harsh skin, may all be the cause of seeking advice; or again it may be a crop of boils, a carbuncle, or some pudendal irritation.

These are indefinite early signs, one or more of which may or may not be present. These symptoms must be described in greater detail; there is no definite line between the ordinary symptom and "complications." One case may be detected early with very slight symptoms another may not come to notice until coma has appeared.

*Polyuria* is a very frequent early symptom. It may appear suddenly in acute cases and cause the patient much distress. Whilst there is often some relation to hyperglycæmia, this is not absolutely true, for the height of the renal threshold and the question of "renal block" will modify the relationship. Generally, the sugar acts as a diuretic, causing an increase in the elimination of water, once the threshold limit is overpassed.

The quantity of urine varies greatly. It may run up to 400 ounces in 24 hours, though amounts ranging from 100 to 200 ounces are much more common.

The urine is generally clear and limpid, with an aromatic odour and sweetish taste. This aromatic odour is generally due to acetone or similar bodies.

The specific gravity is raised, though not invariably so. Generally the urinometer registers from 1,025 to 1,040, but there are rare cases where, with sugar present, 1,014 to 1,016 may be recorded.

With this polyuria, *thirst* is a very frequent symptom, and enormous quantities of fluid may be drunk in the effort to replace the water lost in the urine.

*Increased Appetite or Bulimia* is a very distressing symptom, though it is not very common in the ordinary Bengal case.

The frequent emaciation is due to an insufficient nutrition of the tissues. In severe forms of diabetes the energy requirements may be very considerably increased, even to 20 per cent. more than normal.

These requirements cannot easily be met, for a great deal of the energy contained in the food is excreted by the kidneys as sugar. Although there is a hyperglycæmia, yet the tissues do not receive proper nourishment. It is this tissue starvation that accounts for the emaciation of the severe cases. In slight cases the patient is able by over-eating to supply sufficient food for his tissues and the body weight is maintained.

The various systems are affected in the following ways :—

*Affections of the Nervous System and Special Senses.*—Peripheral neuritis is common in diabetes, as already mentioned. There are numbness, tingling, and cramps—neuralgia occurs, and the burning of the hands and feet already referred to is neuritic in origin. Sciatica, intercostal neuralgia, herpes, falling out of the nails, glossy skin, and perforating ulcer of the foot are all to be seen. The neuritis is perhaps most commonly expressed by absent knee-jerks and cramps in the legs.

A diabetic tabes has been described, though the cases on record seem to be before the days of the Wassermann test.

The mental powers may be considerably diminished, and the patient be unable to carry out his ordinary duties.

Of the special senses, vision is most frequently affected, and cataract and retinitis are the commonest complaints. There may be impaired vision from weakening of the muscles of accommodation, and it is not unusual for patients, in the course of treatment, to find as they improve that their spectacles are useless to them.

Cataract occurs in about 12 per cent. of cases. It is usually bilateral, is of the soft variety in the young, but is of the ordinary variety in old diabetics. No satisfactory explanation of this affection has been given; possibly it is due to a general nutritional weakness.

Diabetic retinitis occurs next in frequency after cataract. Three varieties are described:—

(i) Retinitis centralis punctata diabetica, (ii) Retinitis hæmorrhagica diabetica, (iii) mixed forms.

Diabetic amplyopia due to a central scotoma is not uncommon, and so are various inflammatory affections such as conjunctivitis, keratitis, etc.

*Hearing* is occasionally interfered with, both from otitis media and from inflammatory affections of the outer ear. Mastoid abscesses may develop and true nerve deafness has been known to occur.

*Sexual symptoms* are common. There is loss of sexual desire and power in men, and there may be actual impotence. There is sometimes increased sexual desire and power. Similarly, in women sexual feelings are usually impaired, but they may, especially in elderly females, be much increased. When diabetes occurs during the child-bearing period, amenorrhœa usually results, and the uterus may atrophy. Conception is rare, and, if it do occur, abortion is common. A diabetic mother may bear a healthy child, and has never been known to bear a diabetic one (Fuchter). (This diabetic condition must be distinguished from the mild glycosuria which occasionally occurs in pregnancy.)

*Circulatory and Blood Vascular Systems.*—Actual diabetic endocarditis is not common or characteristic, but a fatty degeneration of the muscle and general flabbiness is common. Heart failure is a common event, and may be associated with dyspnœa, faintness, or angina pectoris.

The facts as to arteriosclerosis are confusing; the condition is described as being common in diabetes, but this is not always the case. My patients have tended to have a subnormal blood pressure rather than an excessive one. The occurrence of hyperglycæmia has already been discussed. There is not infrequently an increase in the fatty substances of the blood and a visible milkiness of the blood serum—lipæmia may result. The cholesterin of the blood serum is increased, particularly in the more severe cases—(*vide* Allen, *A. J. M. S.*, March 1917).

Gangrene is a common occurrence in diabetes. It will be discussed later.

*Respiratory System.*—The two principal affections are tuberculosis and pneumonia, with or without gangrene.

Tuberculosis is generally a late event in diabetes, and is often unsuspected until the tubercle bacillus is actually isolated. The disease has an insidious onset, and often shows little cough or fever. It is a frequent cause of death, and, according to German statistics, occurs in about 40 per cent. of cases. I do not think it is so common as this in India, but when it does appear it is rapidly fatal. Curiously enough, the development of tuberculosis is often a sign for the diminution or disappearance of the glycosuria.

Pneumonia is also quite common, and is frequently complicated with gangrene or abscess of the lung. According to Saundby, pulmonary gangrene occurs more frequently in diabetes than in any other constitutional disease.

*The Urinary System* has been already fully dealt with. The kidneys are often enlarged, and there is a characteristic hyaline degeneration of the epithelial cells of Henle's loop. Casts are rare except at the onset of diabetic coma.

*In the Digestive System* there are several common symptoms. The teeth are frequently loose, and are shed from gingivitis, or atrophy of the gums. Pyorrhœa is common, so too are bleeding and spongy gums. Caries and toothache frequently occur.

The saliva is often diminished, the mouth dry, and the tongue dry and red. Sugar does not occur in the saliva, which tends to be less alkaline than normal. In spite of the enormous quantities of food that are taken the digestion is usually good. Constipation is a frequent and distressing complaint, and is often aggravated by the opium taken by the patient.

The liver may be enlarged and tender.

*The Integumentary System.*—The skin affections in diabetes are numerous and important.

So important are they that, in the East at any rate, one's first instinct on seeing a carbuncle is to examine the urine of the patient.

Boils and carbuncles are extremely frequent incidents probably owing to the lowered resistance to infection shown by the diabetic. They generally occur in the neck, back, or between the shoulders, and the carbuncles sometimes reach an enormous size. The most dangerous are those occurring on the face and scalp, especially those on the upper lip and cheek. The pain and constitutional symptoms are usually severe, and an attack of coma may be precipitated.

Another annoying symptom associated with the skin is *pruritus*. This is due to irritation by the saccharine urine and to the growth of a hyphomycetic fungus in the superficial layers of the skin. This leads to inflammation of the prepuce and glans in the male, and to vulvitis in the female. The irritation caused is sometimes intolerable, especially in women, and may make the patient's life a burden. Like carbuncle, it is a condition in which urinary examination is the first step in treatment. Besides the actual pruritus pudendi, there is often a general itchiness of the skin, probably caused by the circulating sugar. Other skin affections are not uncommon, the chief are purpura, dermatitis herpetiformis, and urticaria. A condition known as diabeté bronzé is an uncommon event; it is associated with hæmachromatosis. Herpes Zoster and perforating ulcer also occur.

*Xanthoma diabeticorum* is a rare condition occurring in diabetes and arising from inflammatory papules. The lesions are small, about the size of a pea, and occur generally on the buttocks, elbows, and knees; they are accompanied by burning, itching, or tenderness.

The condition is a rare one, only forty cases in all having been reported, practically all in men. (Pusey.)

*Diabetic gangrene* is not uncommon. It occurs most commonly in diabetics under fifty, who have not much glycosuria. It appears, as a rule, in the lower limbs, and may be either of the moist or dry variety, most commonly moist. It may occur spontaneously, but frequently is associated with some slight traumatism to tissues already of low vitality. The involved areas may become gangrenous directly, first becoming livid and then dry, grayish, or black. In other cases there may be first the appearance of vesicles or blebs, under which the gangrene occurs.

Sometimes the gangrene is very superficial and in small patches involving little more than the corium, but it may affect a toe, part of the foot, or the whole limb. In cases following wounds or abrasions, which are the most common in races who go nearly barefoot, the gangrene is due to infection in tissues of low vitality, but in spontaneous forms it is due to embolism or endarteritis.

*Acidosis, Ketonuria, and Diabetic Coma.*—No part of work in connection with diabetes is more important than the question of coma and the conditions that lead up to it. Coma is the most frequent cause of death in diabetes, and acidosis and ketonuria are the danger signals.

How important coma is may be judged from Joslin's statistics (*A. J. M. S.*, 1916). He traced 921 of his cases of diabetes, and of this number 425 had died. Of these 425 cases, 273 *or* 64·5 *per cent. died of coma.*

What, then, is this coma, and why is it so fatal? There may be more than one variety of the disease, but there is a special diabetic coma with definite symptoms. This is characterised by a high mortality, by 'air hunger and by the occurrence of acetone bodies in the blood, tissues, urine, and expired air.

Probably the first discovery of acetonuria was that of Peters in 1857, but it was not till 1883 that $\beta$-oxybutyric acid was shown to be the principal organic acid in the urine. Three substances occur in coma; they are diacetic acid, acetone, and oxybutyric acid; when they occur in the urine a condition of *ketonuria* is produced.

Now, though ketonuria occurs in diabetic coma, numerous other conditions, unrelated to diabetes, will cause it, such as fevers, malignant tumours, starvation, and so on.

The most common cause of ketonuria is starvation, whether this be of necessity or from faulty diet in a diabetic. In absolute starvation acetonuria will appear in a few days, and then diacetic and $\beta$-oxybutyric acids occur in the breath and urine. If, however, in place of absolute starvation, a small quantity of carbohydrate be given—not enough to keep up the body weight—then acidosis does not occur.

On the other hand, if large quantities of food be given (sufficient to supply the requisite energy), but no cabohydrates, then acidosis will occur; if the proteins be increased the acidosis will diminish, for the body has the power of forming glucose from protein. If the carbohydrates continue to be excluded from the diet, the acidosis will diminish and possibly entirely disappear.

Thus the acidosis depends upon insufficient carbohydrate, not upon insufficient nutriment.

The amount of ketonuria caused by carbohydrate starvation varies in different people, and as has been already mentioned, other pathological conditions may cause acidosis. Of these chloroform poisoning, continual vomiting, eclampsia, fevers, etc., are the most important, particularly when, as must be often the case, partial starvation accompanies these maladies.

Similarly, in diabetes, there is also a starvation, for though the tissues contain plenty of sugar they are unable to take advantage of it.

It is well known, too, that in diabetes the sudden removal of carbohydrate *alone* from the diet may precipitate acidosis or even an attack of coma, but if there be *complete* starvation—carbohydrates, fats, and proteins—then acidosis does not occur.

As a set-off to this condition we have three others associated with acidosis, namely, the persistent vomiting of children, chloroform poisoning, and phosphorous poisoning. All these conditions are associated with ketonuria, but are not associated with carbohydrate starvation. Most clinicians in the East have had unhappy experiences connected with the administration of anæsthetics to diabetics, with resulting coma. The other conditions are uncommon.

The most important of the ketone group, from a clinical point of view, is diacetic acid. It is the most easily detected, and is in itself an index.

Whilst in acidosis acetone is the first to appear, diacetic acid occurs next, and butyric acid last. Acetone is only of importance from the order of its appearance, for it is questionable whether it occurs in the body, or is only a decomposition product of diacetic acid. In its turn, the diacetic acid is of less significance than the butyric acid, which may largely exceed the other substances. Acetone itself is not easily dealt with in the normal organism, and is not easily excreted through the kidneys; the acetone which appears in the expired air of patients suffering from acidosis possibly derives from the diacetic acid of the blood. Thus, acetone is not likely to be of much importance in the causation of coma, it is rather an indicator of the presence of diacetic and butyric acids.

We must now consider whence these acetone bodies come. It is plain that they must derive from proteins, carbohydrates, or fats. It is not likely that carbohydrates are their source, for we have seen that a sudden stoppage of carbohydrates may cause ketonuria.

It is much more probable that these acetone bodies arise from fats alone, or from fats and proteids. So far as proteids are concerned, Embden's experiments show that certain units of the protein molecule will cause an increase in the diacetic acid. But in similar experiments it has been shown that the ketones formed are greater in amount than the protein ingested, showing that some of them must have come from fats. Probably, in ordinary cases, fat is the chief source, but in severe diabetes some of the ketones derive from proteins as well.

Forsner has shown that, under strict experimental conditions, the administration of fat may increase the acidosis, whilst it is well known that in doubtful cases fats must be given most carefully to diabetics lest acidosis be produced.

It is not necessary here to enter into the chemistry of the fatty acids. It appears (according to Hewlett) that the trouble commences at the stage where $\beta$-oxybutyric acid is formed. Either there is a failure to burn this acid normally and diacetic acid is formed, or else the normal process stops when diacetic acid is produced (Hewlett). Wherever the carbohydrate metabolism is deficient, ketonuria is most common, and this is associated with defective fat combustion.

Possibly the final combustion of fats is assisted by the simultaneous combustion of carbohydrates, or, as it has been said, in the body fats burn in the flame of the carbohydrate metabolism.

But it is also probable that the disturbed carbohydrate metabolism of diabetes is associated with some lessening of the ordinary powers of the system so that it is no longer able to deal with fats, and thus facilitates acidosis.

*The Cause of Coma in Diabetes.*—It has already been shown that, in diabetic coma, the amount of urinary and expiratory acetone are considerably increased ; in the blood the quantity is increased some ten or fifteen times. What then is the rôle of acetone, and what has it to do with coma ? In itself it is a harmless substance, or nearly so, and is not present in sufficient quantity to cause intoxication. It is rather to be considered as an indicator of the amount of diacetic and butyric acids in the system.

We know that during metabolism acids are constantly being formed ; the carbon of proteins, carbohydrates, and fats is largely burnt to $CO_2$ ; the sulphur of proteins is partly converted into sulphuric acid, whilst the phosphorus of nucleo-proteins and lecithin is changed into phosphoric acid. Various organic acids also are formed during the course of these processes, such as the fatty acids from fat, and the organic acids from the deaminization of amino acids. Now, if these processes do not go on to their normal termination, *i.e.*, if the water and carbon-dioxide are not fully formed, then we have an increase of acid in the body, some of which is excreted in the urine. These acids may produce their effects of intoxication either by means of their acid qualities, by means of some specific toxic effect produced by their neutral salts (ketones), or by a combination of the two. Whichever may be the cause, the symptoms are those of a true acid intoxication. There is an increase of urinary ammonia, the alkalinity of the blood is diminished, the carbon dioxide content of the blood is lessened, the alveolar $CO_2$ tension is low, the breathing is typical, and a much larger than normal quantity of alkali must be administered in order to change the reaction of the urine from acid to alkaline.

Regarding the action of the neutral salts (ketones), it has been experimentally shown that the administration of sodium $\beta$-oxybutyrate is followed by symptoms closely resembling diabetic coma.

It is perhaps safer to say that whilst coma results mainly from an acid intoxication, the specific toxic properties of the salts of the ketonic acids may play a part in its pathogenesis. (Hewlett.)

*The Symptoms of Diabetic Coma.*—Generally, these are at first vague and indefinite. The patient is listless, languid, and not quite himself; later, he becomes restless and excited, tossing about in bed. There may be headache and occasional vomiting. Gradually, the symptoms become more marked; the speech becomes thick, or incoherent; dullness increases, and eventually coma supervenes. The pulse becomes small, of low tension, and rapid. But the classical feature is the respiration. A peculiar form of dyspnœa develops, known as "Kussmaul's air hunger." It commences with inspiration, and then affects expiration. The breathing becomes loud and laboured, as if the patient were making some enormous effort; he is in effect breathing hard and deeply in the attempt to get more oxygen into his lungs. With this air hunger there is cyanosis, and a peculiar odour of the urine and breath due to acetone. Albuminuria occurs and enormous numbers of hyaline and granular casts appear in the urine.

The condition is nearly always fatal, generally in two to four days, though occasionally temporary rallies occur under treatment, and in rare instances there may be recovery.

Various other forms of coma have been described in diabetes; they are due rather to some inter-current disease, not to the diabetes.

We have already quoted Joslin's statistics relative to the importance and dangers of coma as a termination of the disease; other observers give similar results. Thus, Nannyn had 19 cases of coma in 44 fatal cases; Frerichs had 150 deaths from coma in 250 fatal cases; Taylor 26 out of 43; and Williamson 28 out of 40.

Apart from the actual chemical changes resulting in coma, there are certain factors which predispose to the condition.

Generally speaking, the diabetic leading an ordinary guarded life has no immediate risk of coma, though it always looms in the distance as a terrible danger. But any sudden indiscretion may be enough to upset the hardly won balance and precipitate a disaster. This indiscretion may be on the part of the patient, or sometimes, most lamentably, on the part of the medical man. Constipation and indigestion are decided dangers, so too is excessive fatigue. The diabetic can take, and is the better for, his regular daily exercise, but let him exceed his limit and the risk of coma threatens him. Acute diseases such as pneumonia are a danger, whilst a carbuncle is always a source of alarm. The administration of anæsthetics to diabetics is a most anxious procedure, and most of us have had unhappy experiences in this respect.

It is probable that it is the anæsthetic rather than the operation which is so dangerous, but in any case, except where there is absolute necessity, operations on diabetics are best left severely alone.

Finally, a not infrequent exciting cause of coma is the sudden withdrawal of carbohydrates whilst leaving the other articles of diet, particularly the fats. Many a diabetic has gone along quite comfortably on a modified diet for many years, until he has had the misfortune to meet an enthusiastic young doctor who persuades him to eliminate his carbohydrates, and dies in coma as a result. This condition only occurs when fats and proteids are left in the diet; complete starvation does not favour coma.

*Urinary Changes Indicative of Acidosis.*—The appearance of a large number of hyaline and granular casts has already been referred to; they appear as coma develops. At this period, too, the urinary sugar may markedly diminish, so that the percentage of $\beta$-oxybutyric acid may be larger than that of the sugar.

*Acetone.*—Qualitative tests.

1. Frowner's test. To 10 c.c. of urine add 1 gramme of solid potassium hydrate. Before solution has occurred add 10 to 12 drops of a solution of salicyl aldehyde in absolute alcohol and warm the mixture to about 70°C. At the zone of contact of the alkali and urine an intense purplish red ring develops in the presence of acetone. This test is specific for acetone, and gives no reaction with

diacetic acid; it is extremely delicate and it may be performed without distilling the urine, as must be done with the older tests. It is recommended for routine use.

2. *Legal's Test for Acetone.*—To 5 c.c. of urine add a small crystal of sodium nitroprusside. After solution has occurred (the urine should be cool) sodium or potassium hydrate is added in excess.

A ruby red colour appears, which fades rapidly to yellow. This colour is given both by creatinin and acetone. Therefore, before the ruby red colour has faded glacial acetic acid is added in excess. In the presence of acetone the red colour will change to a purple or violet tint, while, with creatinin, the red colour will fade at once to a clear yellow or green.

This test is positive with diacetic acid. It will be positive with the untreated urine only when considerable amounts of acetone are present. To render the method more delicate, the distillate from the urine should be used.

*Diacetic acid.*—Qualitative tests.

1. Arnold's test. Two solutions are necessary.

Solution I.

| | | |
|---|---|---|
| Paramidoacetophenon | ... | 1 gram. |
| Distilled water ... | ... | 100 c.c. |
| Concentrated HCl. | ... | 2 c.c. |

The paramidoacetophenon is dissolved in the water with constant shaking and the HCl is added drop by drop till the yellow solution becomes water clear.

Solution II. Sodium nitrite ... 1 per cent. aqueous solution.

To 10 c.c. of Solution I, add 5 c.c. of Solution II and 15 c.c. of urine. Drop in one or two drops of concentrated ammonium hydrate. A non-characteristic brownish red colour appears. Add an excess of strong HCl. If diacetic acid be present a beautiful purple colour developes. In the absence of diacetic acid the red colour changes to yellow on the addition of acid.

*This test is not positive with acetone, with β-oxybutyric acid, nor with drugs.* It is thus much preferable to the ferric chloride test.

2. *Gerhardt's test for diacetic acid.*

To 10 c.c. of urine a ten per cent. solution of ferric chloride is added drop by drop till no further clouding is noted.

The mixture is then filtered. A few drops more of ferric chloride are then added to the filtrate. If diacetic acid be present, the filtrate should now show a claret red precipitate. This colour may be due not to diacetic acid, but to certain drugs many of which are used by some people in the treatment of diabetes and which are certainly in common use. The chief of these are salicylic acid, diuretin, aspirin, salol, phenacetin, acetates and formates. If the solution be boiled, the red colour due to the volatile diacetic acid will fade, whilst that due to all of the above drugs (except the acetates and formates) will remain.

*Oxybutyric Acid, Qualitative Test.*—Hurt's test. This test is not very delicate, but it is the most suitable for clinical work.

To 20 c.c. of urine add an equal quantity of water and a few drops of acetic acid.

The mixture is boiled down to about 10 c.c. in volume (to remove acetone and diacetic acid). The volume is then brought up to 20 c.c. with water and 10 c.c. of the mixture is put in each of two test-tubes. To one of these add 1 c.c. of hydrogen peroxide, warm gently and allow to cool. Then add to each tube 0·5 c.c. of glacial acetic acid and a small crystal of sodium nitroprusside; next float a layer of 2 c.c. of concentrated ammonium hydrate on the solution in each tube and allow the tubes to stand for four or five hours. If $\beta$-oxybutyric acid be present in the urine, the tube to which peroxide was added will show a purplish red contact ring, whilst in the control tube no purple colour will appear.

## CHAPTER IV.

### Prognosis and Treatment.

Before discussing the treatment of diabetes, it is advisable, even at the risk of repeating what has been said before, to investigate the principal causes of death. This is the more necessary, in that, with the introduction of the treatment by alimentary rest, a new era has opened for the diabetic; this is particularly the case in the East.

Fortunately, we have in Joslin's statistics (J.—A. M. S., 1916, p. 317) a detailed analysis of nearly one thousand cases that were under his care. Of a total of 945 cases, 921 were traced, and of these cases 425 have died (the period of enquiry ranged from 1894 to 1915).

The analysis is as follows:—

*Joslin's Table of Causes of Death of Diabetic Patients seen in Private Practice*, 1894—1915.

| | | Number. | In Hospital. | Outside Hospital |
|---|---|---|---|---|
| A. DEATHS WITHOUT COMA | | 147 | 2 | 145 |
| I. Miscellaneous ... ... | | 16 | 0 | 16 |
| Pernicious anæmia ... | 1 | | | |
| Old age ... ... | 2 | | | |
| Diabete ... ... | 8 | | | |
| Cirrhosis ... | 2 | | | |
| Suicide ... ... | 2 | | | |
| Drowned ... ... | | | | |

| | | Number. | In Hospital. | Outside Hospital. |
|---|---|---|---|---|
| II. CANCER | | 17 | 1 | 16 (also 1 with coma). |
| Face | 1 | | | |
| Æsophagus | 1 | | | |
| Stomach | 4 | | | |
| Rectum | 1 | | | |
| Liver | 6 | | | |
| Bladder | 1 | | | |
| Bone | 1 | | | |
| Unknown site | 1 | | | |
| III. TUBERCULOSIS PULMONALIS | | 16 | 1 | 15 (also 5 with coma and 2 with infections.) |
| IV. CARDIO-RENAL AND VASCULAR | | 62 | 0 | 62 |
| Cardiac | 28 | | | |
| Bright's | 14 | | | |
| Cerebral Hæmorrhage | 14 | | | |
| Arterio Sclerosis | 6 | | | |
| V. INFECTIONS | | 36 | 0 | 36 |
| Tonsillitis | 11 | | | |
| Pneumonia | 15 | | | |
| Influenza | 3 | | | |
| Gall-stones | 1 | | | |
| Septic and Gangrenous legs | 9 | | | |
| Carbuncles | 4 | | | |
| Acute Appendicitis | 2 | | | |
| Erysipelas | 1 | | | |
| B. DEATHS WITH COMA | | 273 | 15 | 258 |

If we examine these figures, we find that 64·24 per cent. died in coma. This is perhaps rather lower than the average usually given, but it is evident that coma is by far the most common termination of the disease.

Tubercle gives surprisingly low figures under 4 per cent. According to Joslin we look on tuberculosis in diabetes much too seriously," for when the treatment of diabetes is faithfully carried out, these patients do quite well." This is not in accordance with our experience in India, where pulmonary tuberculosis in the diabetic is looked on as an extremely grave event.

*Heart and Kidney cases* accounted for 62 deaths at an average age of 64 years. It is probable that with improved dietetic methods for treating diabetes these cases will become less frequent.

*Infections* generally lower the tolerance of a diabetic for carbohydrate and thus increase the severity of the disease. "Recent experience with fasting treatment shows that in the presence of an infection a diabetic becomes sugar free very slowly" (Joslin). Whilst this does not agree with my experience in India, undoubtedly the infection is an extra load for the diabetic to carry, and to it he often succumbs. In these American statistics there were but 36 deaths. My impression is that infection is of more importance than this in India, owing to the frequency of abrasions and minor skin affections.

It remains to consider coma.

This is by far the most common cause of death, accounting for 65 per cent. of Joslin's cases. Now, coma, though extremely difficult to treat when once established, is frequently brought on by certain well defined causes, and, if these are guarded against, the onset of coma may be warded off. The chief causes are :—

1. *Anæsthetics.*—Ether or chloroform, like sepsis, are added burdens to the diabetic, and their administration

may be just sufficient to turn the scale, so favouring coma. Whilst one would not lay it down that anæsthetics should not be given to diabetics, yet their administration is certainly attended with risk to the patient and anxiety for the doctor.

2. *Infections* are of great importance, in that they persist for a long time, diminish the patient's general health, and prevent him from obtaining the exercise so necessary to him. The great majority of these infections are at first trivial, but from neglect become more severe and may thus predispose to coma.

3. *Mental excitement* may favour the onset of coma. A sudden fit of temper, some acute business worry, an unexpected bereavement, any of these may excite the dreaded attack of coma.

4. *Kidney disease.*—Patients with this complication are particularly susceptible to coma, for their damaged renal epithelium is unable to carry off the increased quantity of organic acids, and this is particularly the case with elderly people.

5. *Faulty dietary* certainly may cause coma. This is especially seen after a sudden withdrawal of carbohydrates, with a persistence or increase of fats. Strangely enough, the withdrawal of *all* food as exemplified in Allen's treatment does not precipitate a coma; it occurs in those cases where the carbohydrate alone is ruthlessly cut down and the fats and proteins increased.

6. *Rapid loss of Body Fluid.*—Vomiting at the onse of coma usually means death, for the patient is deprived of fluid with which to eliminate acids. So, too, prolonged vomiting may influence an acidosis which in its turn may cause coma.

Joslin gives a further instructive table showing the duration of life of his cases. He says:—"It is obvious that among the living cases will be found many in whom the disease is of recent origin; for this reason the duration of life of the living cases appears far too low in the table."

*Duration of life of* 408 *fatal and* 490 *living cases of diabetes as at*—1-12-1915.

| Age at onset | Number of cases | | Average duration in years | |
|---|---|---|---|---|
| Years. | Fatal. | Living. | Fatal. | Living. |
| 0 to 10 | 33 | 9 | 2·06 | 4·44 |
| 11 to 20 | 48 | 27 | 2·79 | 2·70 |
| 21 to 30 | 40 | 50 | 3·30 | 4·90 |
| 31 to 40 | 53 | 71 | 4·43 | 6·12 |
| 41 to 50 | 71 | 146 | 6·08 | 7·04 |
| 51 to 60 | 97 | 120 | 6·63 | 6·29 |
| 61 to 70 | 52 | 55 | 6·00 | 5·38 |
| 71 to 80 | 14 | 11 | 3·71 | 4·45 |
| 81 to 90 | 0 | 1 | ... | 0·33 |

# CHAPTER V.

## TREATMENT.

THE treatment of diabetes may be said to have passed through three or four stages.

(i) Treatment by drugs alone.

(ii) Treatment by drugs associated with some carbohydrate restriction.

(iii). Treatment by dietetic methods on the Naunyn and Von Noorden plans, *i.e.*, modified diet, oatmeal, green vegetable days, and so on, with drugs only rarely given.

(iv) Treatment by alimentary rest, after the plan devised by Allen.

This method of Allen's is so successful, and has introduced such changes in our conception of the disease, that it has revolutionized the treatment of diabetes all over the world.

It is proposed therefore to describe Allen's method fully in the first instance, and then refer more briefly to other and less successful plans of treatment.

His first paper was published in the *Boston Medical and Surgical Journal* for February 1915, and it is from this article that the description is taken—modified in parts to suit Eastern conditions. Let us refer again to physiology for a moment. Allen's conception of diabetes is that there is a loss of power to utilize sugar, not a mere acceleration of sugar formations. He holds that the internal function of the pancreas makes possible in some way the utilization of sugar by the tissues, but finds no proof that the adrenals oppose this function of the pancreas, or that the pancreas inhibits sugar formation or opposes the adrenals in any way. (These are disputed points, but such are Allen's views). He is rather of opinion that diabetes is first a weakened function of carbohydrate mechanism, subsequently a weakening of protein metabolism, and finally, in the severer cases, imperfect metabolism of fat.

There is experimental support for Allen's theories. He found that on removing nine-tenths of a dog's pancreas, if he tried to keep the dog fat and satisfy its large appetite the animal went steadily down hill and died in extreme cachexia. He further found that he could stop the glycosuria in the dog by making the animal fast and afterwards restricting the food he takes to a low diet, large enough to support life, though not large enough to cause glycosuria.

Allen's method may be described as a means of re-education. Believing that the body has lost (or partially lost) the power of dealing with carbohydrates, the patient is starved until all sugar has left the urine; his metabolic processes are then gradually re-educated to deal with increasing quantities of carbohydrate, &c., until sufficient food is being taken to support life in comfort.

The original description is thus :—" When the fasting patient has been free from sugar for 24 to 48 hours, the next step is to begin feeding slowly and cautiously. There need not be a fixed programme. It is desirable to individualise the diet to suit the needs of different patients, and various physicians may have personal preferences of their own. The one requirement is that the patient must remain free from both glycosuria and acidosis. Any trace of sugar is the signal for a fast day, with or without alcohol. The original fast, to clear up the urine in the first place, may be anything from two to ten days, but after that no fast need be longer than one day. The things to be considered in he diet are carbohydrate, protein, fat and bulk. Frequently the first thing given after the fast is carbohydrate. No distinction is necessary between different forms of starch, but there are advantages in using vegetables, following Joslin's convenient classification on the basis of carbohydrate content. The first day after fasting, the only food may be 200 grams of vegetables of the 5 or 6 per cent. class. This is increased day by day until a trace of glycosuria appears, which is checked by a fast day. The purpose of such a programme is to learn the carbohydrate tolerance, and to clear up the last traces of acidosis. After this carbohydrate period, or sometimes in place of it, protein is given. On the first day perhaps one or two eggs are given, and nothing else. More protein, generally as eggs and meat, is added day by day until the patient either shows glycosuria, or reaches a safe protein ration. The purpose here is to learn the protein tolerance and to cover protein loss as quickly as possible. Fat is somewhat less urgently needed, except in very weak and emaciated patients, and it can be added gradually as conditions seem to indicate. An element of bulk in the diet is necessary to give the comfortable feeling of fulness and to prevent constipation."

Such being the theory of the treatment, how is it to be applied in practice? The most important factor is the intelligence of the patient, coupled, in the case of Hindus, with caste and its restrictions. Whilst there is no one easier to treat on this plan than the educated and middle-aged Bengali Hindu (of the bhadralog class), there are two most difficult classes of patients. One of these is the up-country Hindu, a Vishnuvite and a strict vegetarian, whose main diet consists of hand-ground flour, pulses, and vegetable curry, with sometimes a little goat's flesh, or a duck's egg for the Sivites, or non-vegetarian; the other difficult class is the Marwari Jain, who is, if anything, stricter than the most orthodox Hindustani! Whilst one can sometimes devise a diet that an up-country Hindu can take with mutual satisfaction, it is almost impossible to do so for a Jain. This class are absolute vegetarians will partake of no animal life, and live mainly on flour, rice, pulses, milk and curds, fruit, and sugar. I have succeeded in rendering one such case sugar free for a few weeks on Allen's plan, but had no permanent success.

Wherever it is in any way practicable, the patient should be in hospital or in a nursing home. It is difficult to carry out treatment efficiently in a private house; certainly the patient must be prepared to go to bed for the first few days of the course. The principles of the treatment should be carefully explained and an assurance given that probably a very little fasting will be required. It is here that the patient's intelligence is required. Educated people, as a rule, will see the importance of obeying instructions, but with uneducated agriculturists (Cases III and IV), it is generally impossible to prevent them slipping out of hospital and begging or stealing food.

Before commencing starvation, the patient must be under observation for a few days on ordinary diet, living a quiet uneventful life. The total quantity of urine passed in the 24 hours must be measured, and the sugar estimated. It is necessary to know the total quantity of sugar in grains; two, three or four per cent. of sugar conveys no information, unless one knows the total amount of urine being passed, and a better mental effect is produced if one can tell the patient that the three or four thousand grains of sugar he passed yesterday has dropped to zero to-day!

At the same time, advantage should be taken of the opportunity to make other examinations according to the clinical facilities available. It is rarely possible in the East to work out the D. N. ratio or the respiratory quotient, but there is no difficulty in estimating the blood sugar, the presence or absence of albumen and other abnormalities, the blood pressure, weight, and so on. These should all be done and recorded.

Having obtained these facts, and knowing the average daily excretion of sugar, the patient is put to bed and starvation commenced. The patient is required to stay absolutely in bed, except for his visits to the bath-room.

The bowels should be freely opened, either with castor-oil or salines, preferably castor-oil. This is most important, both to get rid of waste material in the intestine and to diminish the risk of acidosis. Water may be given freely by the mouth, and I generally order a cup of tea or coffee (without milk or sugar) to be given morning and evening. If there be any acidosis, or if the patient be at all pale, alcohol, either whisky or rum, should be given to the extent of four ounces daily, diluted with water and spread over the 24 hours.

Under such a régime, the urine is generally free from sugar (by the Benedict test) in 48 hours. Rarely, a longer fast is required, but, as a rule, 48 hours are sufficient. American observers have fasted their patients for seven to ten days, but that is rarely necessary in India.

At the end of the fast, the patient generally feels a little weak, he may have lost two or three pounds, but he is mentally uplifted at the disappearance of his sugar.

If one is able to make a chart of the sugar excretion, it is extraordinary to note the sudden fall from four or five thousand grains to some four hundred at the end of the first day, and to zero at the end of the second. The graphic method encourages the patient; he can *see* what progress he is making; then too the invalid feels better. The urine diminishes enormously in quantity, and has not to be passed so frequently, thus giving greater rest to the patient. The pruritus and similar annoying symptoms disappear, and a general improvement is noticed.

When the urine is sugar free, we begin to administer carbohydrates as soon as possible, in order to re-educate the organism to deal with them. The carbohydrate is administered in the form of green vegetables, for these have a low carbohydrate content and at the same time are bulky and satisfy the appetite.

Vegetables are divided into four main classes, those that contain 5 per cent. of carbohydrate, 10 per cent., 15 per cent. and 20 per cent. We commence with the 5 per cent. class and gradually work up to the 20 per cent.

In severe cases it may be necessary to prescribe twice boiled or even thrice boiled vegetables. In ordinary cases, the vegetable should be boiled in at least four times its weight of water; after it is fully cooked, it is to be freed from water and weighed, then stewed for 15 minutes in a little clear broth, and flavoured with pepper and salt to taste. When twice or thrice boiled vegetables are ordered, two saucepans are required; when the vegetable is half cooked, it is transferred to the boiling water in the second pan and boiled for 10 minutes. If a third boiling is required the water in the first saucepan is thrown away, fresh water brought to the boil and the vegetable once more transferred. After this it is stewed in broth. The diet table given below is that used by Leyton of the London Hospital (Pract. 16th Nov. 1915). It is suitable for European use in the cold weather within reach of reasonable supplies. Reference to the subsequent tables will show what other vegetable may be substituted from time to time, and an attempt will be made to indicate some hot weather and rainy season vegetables. Local conditions of climate and elevation will necessitate some modification, but the tables will show the lines along which one should work.

*Diet for the* FIRST DAY AFTER THE URINE IS FREE FROM SUGAR:—

Saline in the morning.

*Breakfast.* Cup of tea or coffee without milk or sugar: 70 grams of French beans or asparagus, or seakale prepared as directed. C. H. 1·5. Pr. ·6. Fat ·8.

*Lunch.* Cup of tea or coffee, no milk or sugar. 80 grams French beans or asparagus or seakale. C.H. 2. Pr. ·8. Fat 1·1.

*Tea.* Cup of tea, no milk or sugar.

*Dinner.* Cup of tea or coffee. 70 grams cooked French beans or asparagus. C. H. 1·5. Pr. ·6. Fat ·8.

Carbohydrate 5 grams. Protein 2 grams. Fat 2·7 grams. Calories 52.

(NOTE: A bean resembling French bean is obtainable in Bengal for quite two-thirds of the year. Spinach or one of the many varieties of "Sag" may be substituted, or *thrice boiled* cabbage.)

SECOND DAY AFTER THE URINE IS FREE FROM SUGAR.

Saline in the morning.

*Breakfast.* Large cup of tea or coffee, no milk or sugar. 200 grams cooked French beans. C.H. 3. Pr. 1·2. Fat 1·6.

*Lunch.* 200 grams cooked French beans or seakale or asparagus. C. H. 4. Pr. 1·26. Fat 2·2.

*Tea.* Large cup of tea, no milk or sugar.

*Dinner.* Cup of tea or coffee. Cooked French beans or asparagus 200 grams. C. H. 3. Pr. 1·2. Fat 1·6. Carbohydrate 10 grams. Protein 4. Fat 5·4. Calories 104.

Third Day after urine is free erom sugar.

Saline in the morning.

*Breakfast.* Large cup of tea or coffee. Cream 5 c.c. 1 boiled egg. 7 oz. raw lettuce. C.H. 6. Pr. 8·4. Fat 6·1.

*Lunch.* 1 egg, 200 grams of cooked cucumber. C.H. 6. Pr. 7·6. Fat 5·4.

*Tea.* Cup of weak tea with 5 c.c. of cream.

*Dinner.* 200 grams cooked French beans; 1 egg. C.H. 3. Pr. 7·2. Fat 6·6. Carbohydrate 15. Protein 24. Fat 19. Calories 327.

Fourth Day after the urine is free from sugar.

Saline in the morning.

*Breakfast.* Large cup of tea or coffee. 10 c.c. of cream. 1 boiled egg. 200 grams of raw lettuce. C. H. 6. Pr. 8·4. Fat 5·7.

*Lunch.* 200 grams cooked cabbage; 1 boiled egg. C.H. 11·2. Pr. 9. Fat 5·6.

*Tea.* Cup of tea. 10 c.c. cream. Fat 1.

*Dinner.* 200 grams cooked French beans. 1 egg. C.H. 4. Pr. 7·6. Fat 7·2. Carbohydrate 21. Protein 2. Fat 20. Calories 360.

Fifth Day after urine is sugar free.

| Saline in the morning. | | CH. | Pr. | Fat. |
|---|---|---|---|---|
| *Breakfast.* Coffee, large cup. Cream 10 c.c. | | | | 1 |
| One egg ... ... ... | | | 6· | 5 |
| Raw lettuce ... | 200 grams ... | 6 | 2·4 | ·6 |
| *Lunch.* Cooked lean meat | 30 grams ... | | 8· | 3 |
| Cooked cabbage | 200 grams ... | 11·2. | 3 | ·6 |

| | C.h. | Pr. | Fat. |
|---|---|---|---|
| *Tea.*—Tea 200 c.c. Cream 10 c.c. | 0 | 0 | 1 |
| One egg | 0 | 6 | 5 |
| *Dinner.*—Clear broth 250 c.c. | 0 | 5 | 0 |
| One egg | 0 | 6 | 5 |
| Cooked French beans, seakale or asparagus 200 grams | 4 | 1·6 | 2·2 |

### SIXTH DAY AFTER URINE IS SUGAR FREE.

Saline in the morning.

| | | | |
|---|---|---|---|
| *Breakfast.*—Thin cream 10 c.c. | 0 | 0 | 1 |
| Coffee<br>Tea 200 c.c. One egg | 0 | 6 | 5 |
| Raw lettuce 200 c.c. | 6 | 2 | 1 |
| *Lunch.*—Cooked lean meat 100 grams | 0 | 24 | 9 |
| Boiled potatoes 50 grams | 10 | 1 | 0 |
| *Tea.*—Tea 200 c.c. Cream 10 c.c. | 0 | 0 | 1 |
| *Dinner.*—One egg | 0 | 6 | 5 |
| Cooked cabbage 100 grams | 6 | 2 | 0 |
| French beans or seakale 100 grams | 2 | 1 | 1 |
| | 24 | 42 | 23 |

Calories 565.

### SEVENTH DAY AFTER URINE IS SUGAR FREE.

Saline in the morning.

| | |
|---|---|
| *Breakfast.*—Coffee or tea | 200 c.c. |
| Clear Broth | 100 c.c. |

| | |
|---|---|
| *Lunch.*—Coffee or tea | 200 c.c. |
| Clear broth | 100 c.c. |
| *Tea.*—Tea | 200 c.c. |
| *Dinner.*—Tea or coffee | 200 c.c. |
| Clear broth | 100 c.c. |

### Eighth Day after Urine is Sugar Free.

Saline in the morning.

| | C.h. | Pr. | Fat. |
|---|---|---|---|
| *Breakfast.*—Coffee or tea 200 c.c. | | | |
| Cream 30 c.c. ... ... | 0 | 0 | 3 |
| One egg ... ... | 0 | 6 | 5 |
| Cooked fat bacon 20 grams ... | 0 | 3 | 10 |
| Raw lettuce 200 grams ... | 6 | 2 | 1 |
| *Lunch.*—Cooked lean meat 60 grams ... | 0 | 16 | 6 |
| Cooked French beans or asparagus 100 grams ... ... | 2 | 1 | 1 |
| Boiled potatoes 50 grams ... | 10 | 1 | 0 |
| *Tea.*—Weak tea 200 c.c. Cream 30 c.c. | 0 | 0 | 3 |
| One egg ... ... | 0 | 6 | 5 |
| *Dinner.*—Clear broth 250 c.c. ... | 0 | 5 | 0 |
| One egg ... ... | 0 | 6 | 5 |
| Cooked cabbage 100 grams ... | 6 | 2 | 0 |
| Seakale, beans or asparagus 100 grams ... ... | 2 | 1 | 1 |
| | 26 | 49 | 40 |

Calories 660.

### NINTH DAY AFTER URINE IS SUGAR FREE.

Saline in the morning.

| | C.h. | Pr. | Fat. |
|---|---|---|---|
| *Breakfast.*—Tea or coffee 200 c.c. Cream 10 c.c. ... ... | 0 | 0 | 3 |
| One egg. Cooked fat bacon 30 grams ... ... | 0 | 11 | 20 |
| Raw lettuce 200 grams ... | 6 | 2 | 1 |
| *Lunch.*—Cooked lean meat 50 grams, Fat 15 grams ... ... | 0 | 16 | 21 |
| Cooked beans or seakale 100 grams ... ... | 2 | 1 | 1 |
| *Tea.*—Weak tea 200 c.c. Cream 10 c.c. | 0 | 0 | 3 |
| One egg ... ... | 0 | 6 | 5 |
| *Dinner.*—Clear broth 250 c.c. ... | 0 | 5 | 0 |
| One egg. Cabbage 120 grams ... | 7 | 8 | 5 |
| Cooked beans or seakale 80 grams | 2 | 1 | 1 |
| | 17 | 50 | 60 |

Calories 952.

### TENTH DAY AFTER URINE IS SUGAR FREE.

Add 30 grams of fat as butter, distribute this amongst the vegetables.

### ELEVENTH DAY AFTER URINE IS SUGAR FREE.

| | C.h. | Pr. | Fat. |
|---|---|---|---|
| *Breakfast.*—Tea or coffee 250 c.c. Cream 30 c.c. | 0 | 0 | 3 |
| One egg. Cooked fat bacon 30 grams | 0 | 11 | 20 |
| Raw lettuce 200 grams | 6 | 2 | 1 |
| *Lunch.*—Cooked lean meat 60 grams | 0 | 16 | 6 |
| Fat 15 grams | 0 | 0 | 12 |
| Cooked beans, asparagus or seakale 100 grams | 2 | 1 | 1 |
| Boiled potatoes 50 grams | 10 | 1 | 0 |
| Butter 30 grams | 0 | 0 | 25 |
| *Tea.*—Tea 250 c.c. Cream 30 c.c. | 0 | 0 | 3 |
| One egg. Boiled potatoes 50 grams | 10 | 7 | 5 |
| Butter 15 grams. | | | |
| *Dinner.*—Clear broth 250 c.c. | 0 | 5 | 0 |
| Cooked cabbage 100 grams | 6 | 2 | 0 |
| Cooked seakale or beans 100 grams | 2 | 1 | 1 |
| Butter 30 grams | 0 | 0 | 25 |
| | 36 | 46 | 102 |

Calories 1,432.

### TWELFTH DAY.

Add fat 30 grams.

Calories 1,702.

### THIRTEENTH DAY AFTER URINE IS SUGAR FREE.

| | C.h. | Pr. | Fat. |
|---|---|---|---|
| *Breakfast.*—Coffee or tea 250 c.c. Cream 30 c.c. | 0 | 0 | 3 |
| One egg. Cooked fat bacon 30 grams | 0 | 11 | 20 |
| Raw lettuce 100 grams | 3 | 1 | 0 |
| White bread 15 grams | 9 | 1 | 0 |
| Butter 15 grams | 0 | 0 | 12 |
| *Lunch.*—Cooked lean meat 60 grams. Fat 30 grams | 0 | 16 | 31 |
| Cooked beans or asparagus 100 grams | 2 | 1 | 1 |
| Boiled potatoes 50 grams. Butter 30 grams | 10 | 1 | 25 |
| *Tea.*—Tea 250 c.c. Cream 30 c.c. | 0 | 0 | 3 |
| White bread 15 grams. One egg | 9 | 7 | 5 |
| Butter 30 grams | 0 | 0 | 25 |
| *Dinner.*—Clear broth 250 c.c. | 0 | 5 | 0 |
| One egg. Cooked cabbage 100 grams | 6 | 8 | 5 |
| Cooked seakale or beans 100 grams | 2 | 1 | 1 |
| Butter 60 grams | 0 | 0 | 50 |
| | 41 | 52 | 181 |

Calories 2,001

### FOURTEENTH DAY.

No carbohydrates and only half quantities of protein and fat (patient may take less but not more than this).

### FIFTEENTH DAY AFTER URINE IS SUGAR FREE.

| | | C.h. | Pr. | Fat. |
|---|---|---|---|---|
| *Breakfast.*—Coffee 250 c.c. Cream 30 c.c. | | 0 | 0 | 3 |
| One egg | | 0 | 6 | 5 |
| Cooked fat bacon | 30 grams | 0 | 5 | 15 |
| Raw lettuce | 100 grams | 3 | 1 | 0 |
| White bread | 15 grams | 9 | 1 | 0 |
| Butter | 15 grams | 0 | 0 | 12 |
| *Lunch.*—Lean cooked meat | 60 grams | 0 | 16 | 6 |
| Fat | 60 grams | 0 | 0 | |
| Cooked beans or seakale | 100 grams | 2 | 1 | 1 |
| Boiled potatoes | 50 grams | 10 | 1 | 0 |
| Butter | 30 grams | 0 | 0 | 25 |
| *Tea.*—Tea 250 c.c. Cream 30 c.c. | | 0 | 0 | 3 |
| White bread | 15 grams | 9 | 1 | 0 |
| One egg | | 0 | 6 | 5 |
| Butter, | 30 grams | 0 | 0 | 25 |
| *Dinner.*—Clear broth 250 c.c. | | 0 | 5 | 0 |
| One egg | | 0 | 6 | 5 |
| Cooked cabbage | 100 grams | 6 | 2 | 0 |
| Cooked French beans or asparagus | 100 grams | 2 | 1 | 1 |
| Butter | 60 grams | 0 | 0 | 50 |
| | | 41 | 52 | 206 |

Calories 2,226

If no sugar appears in the urine, increase the carbohydrate by 5 grams upon alternate days and decrease fat by 5 grams every fourth day.

This diet is at first monotonous, but with a little ingenuity it can be varied considerably. It is devised for use in Europe and, therefore, may require modifications in the East. For example, the Indian egg is much smaller than the English one, so two or even three may be required in place of one. Spinach, cucumber, patal, the various pumpkins are all of low carbohydrate content and can be used in the hot weather when lettuce is unobtainable. Boiled celery is quite appetising—potted meat or anchovy paste may be used for flavouring. Ghi may replace butter when necessary ; mustard oil may also be substituted if preferred. The carbohydrate must be evenly distributed over the four meals, so that the strain on the metabolism may be diffused as much as possible. There are two traditions that are extremely hard to break away from, both for patient and doctor. The first is the desire to increase the protein ration ; this, I suppose, is a legacy from earlier days. One has to remember that the diabetic makes sugar from protein, and that one is trying to re-educate him to deal with carbohydrates. It is the carbohydrates that one has to increase to the limit of the patient's toleration, not the proteins.

The second tradition is connected with the patient's weight. There must be some loss of weight at first generally it is only two or three pounds. Now this is of no importance. It is the presence or absence of urinary sugar that counts, not the loss of a few pounds in weight. It must be remembered that diabetes is a disease of metabolism, and that we cannot afford to put a strain on any part of the organism. It is better therefore to slightly underfeed and keep the patient's weight slightly below normal, but the urine sugar free, than to give food up to the limit and have a constant strain—which may at any moment be a breaking strain—in the metabolic mechanism.

Many tables of carbohydrate equivalents have been worked out. This is one given by Leyton.

Five grams of carbohydrate may be taken as the equivalent of :—

| | | | |
|---|---|---|---|
| 250 | grams | ... | Cooked French beans. |
| 250 | ,, | ... | Boiled seakale. |
| 250 | ,, | ... | Asparagus. |
| 200 | ,, | ... | Spinach (sag, &c.). |
| 200 | ,, | ... | Raw lettuce. |
| 150 | ,, | ... | Raw cucumber. |
| 150 | ,, | ... | Raw celery. |
| 120 | ,, | ... | Steamed Brussels sprouts. |
| 100 | ,, | ... | Steamed cauliflower. |
| 100 | ,, | ... | Stewed cabbage. |
| 90 | ,, | ... | Leeks. |
| 90 | ,, | ... | Raw radishes (moulli). |
| 90 | ,, | ... | Steamed turnip tops. |
| 60 | ,, | ... | Boiled beetroot. |
| 30 | ,, | ... | Boiled green peas. |
| 30 | ,, | ... | Stewed artichokes. |
| 25 | ,, | ... | Boiled potatoes. |
| 7 | ,, | ... | White bread. |
| 7 | ,, | ... | Dry oatmeal. |
| 7 | ,, | ... | Sponge cake. |
| 40 | ,, | ... | Peaches. |
| 40 | ,, | ... | Oranges. |
| 30 | ,, | ... | Mango. |
| 20 | ,, | ... | Plantain. |
| 75 | c.c. | ... | Milk. |

Of the vegetables available in the rains, patal, jhinga, snake-plant and pumpkin are all of about the same value as the cucumber. Tomatoes, kohlrabi, and brinjal are all in the 5 per cent. class.—(Joslin).

What should be done if and when sugar re-appears? In some of the mild cases we see in Bengal sugar doesn't re-appear, except when actual cane sugar is taken in the dietary. Generally, however, it appears sooner or later, and one has to decide what to do.

(*a*) It may appear when the patient has reached a standard diet on which he is supposed to be in equilibrium. Then one should suspect a breach of the rules or adulteration of the food. Possibly, he has taken a large quantity of protein, he may have had rice or sweetmeat, or, as occurred in one case, the milk was being adulterated with cane sugar by the milkman! Again, worry, emotion, an argument with a refractory servant, may all cause a temporary return of sugar.

(*b*) If sugar returns after the fifteenth day whilst the carbohydrate is being increased, but the protein and fats are constant, it is probable that the carbohydrate limit has been passed. The patient must fast until sugar free, then go on half rations and then gradually increase the protein until 1 gram per kilogram is being taken. The fat is to be increased until the requisite amount of energy is in the food and then the carbohydrate is increased 5 grams a week until two-thirds of the amount which led to the appearance of sugar are being taken. This diet should not be added to for several months.

(*c*) If sugar returns between the sixth and the fifteenth day, it is probably due to an excess of carbohydrate.

The patient starves until sugar free and then the diet is increased at double the rate indicated in the chart, but the carbohydrate is kept at one-half of that given when the sugar reappeared. The protein is increased gradually up to 1 gram per kilogram, and so too is the fat, until the necessary amount of energy is being given. The carbohydrate is increased by 5 grams weekly, usually giving the increased amount just before the weekly fast. When the carbohydrate limit is found, the quantity is kept at two-thirds of this for several months.

(*d*) The most difficult cases are those in which sugar returns early, say at the end of the first week. Such patients are severe cases, irritable, and difficult to manage. Constant changes have to be made in the diet and considerable ingenuity exercised until the carbohydrate and protein limit is determined. Fortunately we see few of these cases in the East.

It is of little moment in what form the various foods are given. It may be either 7 grams of white bread or 250 grams of cooked French beans—in each case the equivalent is 5 grams of carbohydrate. But the patient will feel much more satisfied after the beans than after the bread. Alcohol is generally advisable, particularly if the patient is accustomed to it; it prevents acidosis and does the patient good mentally. He feels "that his strength is being supported." There is no virtue in oatmeal or in diabetic foods; the latter are expensive, nasty, and generally useless. Green vegetables, from the salts they contain, are of distinct value and one sort or another can generally be obtained in the plains.

It is essential that the patient should return at regular intervals for examination, and that he should be strictly instructed as to his diet, for it is probable that a patient who commences the Allen treatment and then relapses is worse off than if he had not had the treatment at all.

Even after leaving immediate medical supervision, the patient should measure his daily urine and test it daily with Benedict's solution. In this way only can an effective watch be kept on the progress of the disease. Even if there be no sugar, the weekly fast or semi-fast day must be continued. There must be no worrying about loss of weight; as a rule this will remain below normal, or a few ounces weekly may be gained.

Exercise, if moderate in amount, is decidedly beneficial, but it must not be carried to the verge of exhaustion; it is probable that moderate exercise favours an increased assimilation of carbohydrate.

I have never found it necessary to stop the fasting on account of any intercurrent symptoms. Diarrhœa, excessive acidosis, mental symptoms are instanced by Leyton as possible reasons for stopping the fast. Sometimes, according to Leyton and Cammidge, it may be necessary to stop the fasting for a day or two, before the sugar has absolutely gone, feed lightly for two or three days and then resume the fast until sugar free. Gangrene and septic conditions are no contra-indication to treatment. A case recently occurred of a man who entered hospital for cellulitis of the foot. In due course his urine was examined and found to be loaded with sugar. He was passing 3,500 grains of sugar per diem and 125 ounces of urine. In a day or two gangrene of two toes appeared. With some trepidation he was fasted for 48 hours. The sugar disappeared and the man expressed himself as better. Graduated diet with alcohol was given, and no sign of coma appeared. There was no improvement in the septic foot however, and one morning I found to my dismay that an energetic house surgeon had administered chloroform, removed the gangrenous toes, and made numerous incisions into the foot. Even this caused no coma and no return of sugar. The septic condition progressed, and the patient refusing any further treatment died in three days from septicæmia, but without sugar or coma.

The experience of Cammidge with the Allen treatment confirms that of Leyton. (Pract. Novr. 10, 1916, p. 417.) He emphasises the importance of keeping the fat content of the diet at a low level until a fair tolerance has been obtained for carbohydrate, and so diminishing the risk of acidosis. He advises black coffee and *China* tea, but avoids cream, as it may prolong the fast and increase the risk of acidosis. Like Allen and Leyton, Cammidge is not deterred by some acidosis from continuing the fast. In most cases he gives 1 c.c. of whiskey per kilo of body weight, and endeavours to accelerate the vegetable (carbohydrate ?) diet.

"Unless the acidosis assumes alarming proportions, it is not advisable to discontinue the fast simply because it is increasing, for, as soon as a vegetable diet can be begun, it will probably diminish and steadily fall as the quantity of vegetable is increased. That this improvement is not entirely, or chiefly, due to the carbohydrate contained in the vegetables is shown by the fact that it occurs in patients with a very low tolerance, and also that it takes place before vegetables containing more than a small amount of carbohydrate have been included in the diet.

"My own observations suggest that it is largely dependent upon the potential alkalinity of the vegetables and the physiological proportion of the bases, particularly calcium and magnesium, that they furnish to the tissues. Both calcium and magnesium appear to be important factors in controlling the condition of the heart and nervous system and in warding off coma."

None of my cases treated during the past few months have shewn any signs of acidosis, either clinically or chemically.

The following table is given by Cammidge as an example of the carbohydrate and protein value of different foods. He takes an "average helping" and determines the amount of carbohydrate, etc., in each :—

## PROTEIN FOODS.

*In average servings.*

### GROUP I.

| Oz. | | Protein Grams. | Fat Grams. |
|---|---|---|---|
| | Yolk of egg 2, average (English) ... ... | 5 | 10 |
| 1 | Bacon, average ... ... | 5 | 15 |

### GROUP II.

| Oz. | | Protein Grams. | Fat Grams. |
|---|---|---|---|
| 3 | Sprats ... ... | 9 | 4 |
| 1½ | Ham, boiled ... ... | 9 | 10 |
| 1½ | Sardines ... ... | 10 | 8 |

## GROUP III.

| Oz. | | Protein Grams. | Fat Grams. |
|---|---|---|---|
| 3¼ | Plaice ... ... | 14 | Trace. |
| 3 | Mackerel ... ... | 14 | 6 |
| 2½ | Salmon ... ... | 14 | 7 |
| 2¼ | Tongue, tinned ... | 14 | 15 |
| 3¼ | Whiting ... ... | 15 | Trace. |
| 3¾ | Sole ... ... | 15 | 1 |
| 1¾ | Chicken ... ... | 16 | 2 |

## GROUP IV.

| Oz. | | Protein Grams. | Fat Grams. |
|---|---|---|---|
| 3¾ | Cod ... ... | 20 | Trace. |
| 3¼ | Haddock (fresh) ... | 20 | Trace. |
| 2¼ | Veal ... ... | 20 | 1 |
| 2½ | Mutton (boiled) ... | 20 | 3 |
| 2 | Turkey (light meats) ... | 20 | 3 |
| 2¾ | Duck ... ... | 20 | 7 |
| 2¾ | Herring ... ... | 20 | 7 |
| 2½ | Beef roast ... ... | 20 | 11 |
| 3½ | Lamb roast ... ... | 20 | 13 |
| ... | Eggs 3, average English ... | 20 | 16 |
| 2¾ | Mutton roast ... ... | 20 | 18 |

## CARBOHYDRATE FOODS.

*In average helpings.*

### GROUP I.

| Oz. | | Carbohydrate Grams. |
|---|---|---|
| | ***Boiled Vegetables.*** | |
| 7 | Vegetable Marrow, Pumpkin, Squash, &c. | 0·4 |
| 5 | Seakale | 0·5 |
| 6 | Cabbage | 0·7 |
| 6 | Cauliflower | 0·7 |
| 5 | Lettuce | 0·8 |
| 5 | Turnip | 1·0 |
| 4 | Celery | 1·0 |
| | ***Raw Vegetables.*** | |
| 2 | Lettuce | 1·5 |
| 2 | Endive | 1·5 |
| 2 | Cucumber | 1·8 |

### GROUP II.

| Oz. | | Carbohydrate Grams. |
|---|---|---|
| | ***Raw Vegetables.*** | |
| 1 | Radishes | 2·0 |
| 4 | Celery | 3·5 |
| 1 | Onion | 3· |
| | ***Cooked Vegetables.*** | |
| 3 | Spinach, Sags, &c. | 2·5 |
| 3½ | French Beans | 3·0 |
| 6 | Parsnip | 3·0 |
| 2½ | Onion | 3·5 |
| 4 | Asparagus | 3·6 |
| 4½ | Leeks | 3·8 |

## GROUP III.

| Oz. | | Carbohydrate Grams. |
|---|---|---|
| | *Raw.* | |
| 3½ | Tomato ... ... | 4·0 |
| 2 | Mushroom ... ... | 4·0 |
| | *Boiled.* | |
| 7 | Greens ... ... | 4·0 |
| 4 | Brussels Sprouts ... | 4·0 |
| 4 | Carrots ... ... | 4·0 |
| 2 | Beetroot ... ... | 5·0 |
| | Fruits (as purchased). | |
| 2½ | Lemon ... ... | 4·0 |
| 1½ | Lemon Juice ... ... | 4·0 |
| 2 | Strawberries ... ... | 4·3 |
| 3 | Peach ... ... | 5·0 |
| 1½ | Walnuts (edible part) ... | 4·0 |
| 1 | Almonds ... ... | 5·0 |

## GROUP IV.

| | *Boiled Vegetables.* | |
|---|---|---|
| 6 | French Artichoke ... | 8·0 |
| 3 | Green Peas ... ... | 13·0 |
| | Fruits (as purchased). | |
| 3 | Fresh Pineapple ... | 9·0 |
| 2 | Apricots ... ... | 14·0 |
| 3 | Cherries ... ... | 14·0 |
| 1 | Cocoanut ... ... | 8·0 |
| 1 | Chestnut ... ... | 12·0 |

## GROUP V.

| Oz. | | Carbohydrate Grams. |
|---|---|---|
| | *Fruit (as purchased.)* | |
| 4 | Grapes | 17·0 |
| 10½ | Water-melon | 20·0 |
| 6 | Orange | 20·0 |
| 5 | Apple | 20·0 |
| 4 | Plums | 22·0 |
| 5 | Pear | 22·0 |
| 7 | Plantain (Banana) | 44·0 |
| | *Starchy foods.* | |
| 1 | Bread, average | 15·0 |
| 3 | Hominy, boiled | 15·3 |
| 4¾ | Oatmeal Porridge | 15·6 |
| 3½ | Macaroni, boiled | 15·8 |
| 2¼ | Rice, boiled | 15·8 |
| 2 | Potato chips | 27·0 |
| 5¼ | ,, boiled | 31·0 |
| 4½ | ,, baked | 32·0 |

### *Miscellaneous.*

| | | Protein Grams. | C. Hydr Grams. |
|---|---|---|---|
| 3½ | Lobster (Prawn ?) | 18·0 | 0·6 |
| ¾ | Cream (average) | 0·5 | 1·0 |
| 1 | Milk | 1·0 | 1·4 |
| 1 | Pickles, mixed | ... | 1·0 |
| 2½ | Cheddar Cheese | 20·0 | 3·0 |

McCay (Protein Element, etc., and Scientific Memoirs) gives the following details of foodstuffs more particularly used in the East:—

| | Water per cent. | Protein per cent. | C. H. per cent. | Fat per cent. | Ash per cent. |
|---|---|---|---|---|---|
| Goat's flesh ... | ...... | 24·06 | ...... | 2·50 | 1·10 |
| Fish ... ... | ...... | 17·80 | ...... | 5·04 | ...... |
| Burma rice ... | 11·13 | 6·95 | 77·25 | 0·96 | 1·34 |
| Country rice ... | 11·05 | 6·62 | 81·07 | 0·50 | 1·04 |
| Wheat flour ... | 11·83 | 11·47 | 70·90 | 2·04 | 3·14 |
| Maize ... ... | 11·50 | 9·52 | 68·90 | 4·44 | 3·75 |
| Mung dâl ... | 10·87 | 23·62 | 53·45 | 2·69 | 3·57 |
| Masûr dâl ... | 10·23 | 25·47 | 55·03 | 3·00 | 3·33 |
| Arhar dâl ... | 10·08 | 21·70 | 54·06 | 2·50 | 5·50 |

Joslin of Boston, quoted by Barker (Mon. Med., Vol. IV, p. 833) gives a similar satisfactory account of Allen's fasting treatment. He quotes a series of rules and gives a list of foods arranged according to their percentage of carbohydrates.

*Joslin's rules for tolerance testing in diabetes fasting.*—Fast until sugar free. Drink water freely, and tea, coffee and clear meat-broth as desired. In very severe, long standing and complicated cases, without otherwise changing habits or diet, omit fat, after two days omit protein and halve carbohydrate daily to 10 grams, then fast.

*Carbohydrate tolerance.*—When the 24-hour urine is sugar free, add 150 grams of 5 per cent. vegetables and continue to add 5 grams carbohydrate daily up to 20 and then 5 grams every other day, passing successively upward through the 5, 10, and 15 per cent. vegetables, 5 and 10 per cent. fruits, potato, and oatmeal to bread, unless sugar appears, or the tolerance reaches 3 grams carbohydrate per kilogram of body weight.

*Protein tolerance.*—When the urine has been sugar free for 2 days, add 20 grams protein (3 English eggs) and thereafter 15 grams protein daily in the form of meat until the patient is receiving 1 gram protein per kilogram body weight, or if the carbohydrate tolerance is zero, only ¾ gram per kilogram body weight.

*Fat tolerance.*—While testing the protein tolerance a small quantity of fat is included to the eggs and meat given. Add no more fat until the protein reaches 1 gram per kilogram (unless the protein tolerance is below this figure but then add 25 grams daily until the patient ceases to lose weight or receives not over 40 calories per kilogram body weight.

*The appearance of sugar.*—The return of sugar demands fasting for 24 hours or until sugar free. The diet is then increased twice as rapidly as before, but the carbohydrate should not exceed half the former tolerance until the urine has been sugar free for two weeks, and it should not then be increased more than 5 grams per week.

*Weekly fast days.*—Whenever the tolerance is less than 20 grams carbohydrate, fasting should be practised one day in seven; when the tolerance is between 20 and 50 grams carbohydrate, upon the weekly fast day 5 per cent. vegetables and one-half the usual quantity of protein and fat are allowed; when the tolerance is between 50 and 100 grams carbohydrate, the 10 and 15 per cent. vegetables are added as well. If the tolerance is more than 100 grams carbohydrate, upon weekly fast days the carbohydrate should be halved.

1 gram protein = 4 calories.
1 ,, carbohydrate = 4 ,,
1 ,, fat = 9 ,,
1 ,, alcohol = 7 ,,
6·25 ,, protein contain = 1 gram nitrogen.
1 kilogram = 2·2 pound.
30 grams on cubic centemetres = 1 ounce.

A patient "at rest" requires 25 to 30 calories per kilogram of body weight.

*Strict diet.*—Meats, fish, broths, gelatine, eggs, butter (*ghi*) olive oil, coffee, tea.

*Foods arranged approximately according to percentage of carbohydrates :—*

Reckon the available carbohydrates in vegetables of the 5 per cent. group as 3 per cent., of 10 per cent. group as 6 per cent.

*Vegetables 5 per cent.*

Lettuce
Spinach
Rhubarb
Brinjals
Leeks
Beet, green tops
French Beans
Brussels Sprouts.
Sorrel
Spinach (Sags).
Vegetable Marrow.

Cauliflower.
Tomatoes.
Beans.
Celery.
Asparagus.
Water Cress.
Cabbage.
Radishes.
Pumpkin.
Kohlrabi.
Seakale.

10 *per cent.*
Onions ... ... Squash.
Turnips ... ... Carrots.
Mushrooms ... ... Beet.
15 *per cent.*
Green Peas ... ... Artichokes.
Parsnips ... ... Tinned Lima Beans.
20 *per cent.*
Potatoes ... ... Haricot Beans.
Green corn (Bhoota) ... Boiled Rice.
Boiled Macaroni.

*Fruits* 5 *per cent.*

Ripe Olives (20 per cent. fat).
Grape Fruit.
10 *per cent.*
Lemons ... ... Oranges.
Cranberries ... ... Strawberries.
Blackberries ... ... Gooseberries.
Peaches ... ... Pineapple.
Watermelon.
15 *per cent.*
Apples ... ... Pears.
Apricots ... ... Cherries.
Currants ... ... Mangoes (?)
20 *per cent.*
Plums ... ... Bananas.

Nuts.
10 *per cent.*

Brazil Nuts ... ... Black Walnuts.
Hickory Nuts ... ... Filberts.

15 *per cent.*

Almonds.
Pistachios.
Walnuts.
Pine Nuts.

20 *per cent.*

Pea Nuts.
Ground Nuts.

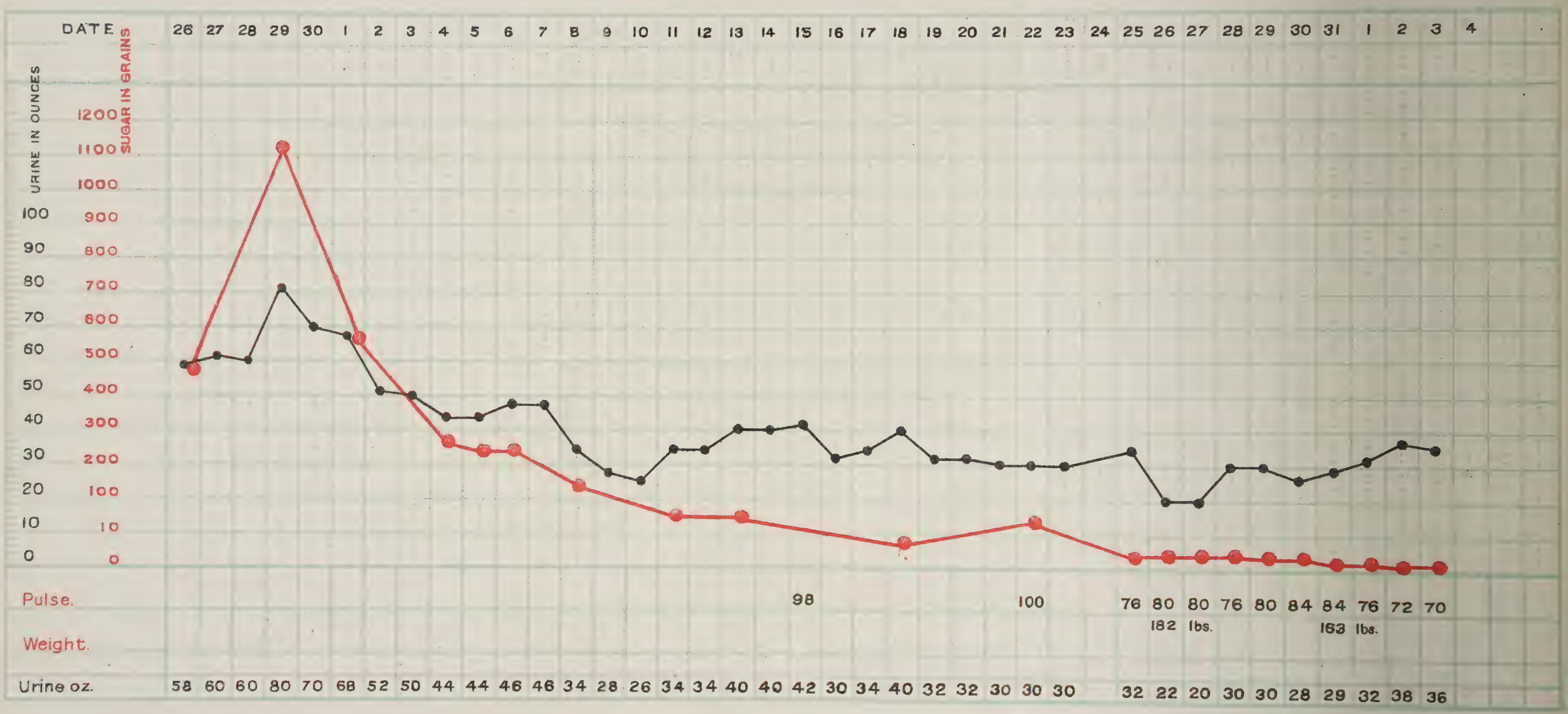
40 *per cent.*

Chestnuts.

30 GRAMS OR ONE OUNCE CONTAIN APPROXIMATELY:—

| | Protein. Grams. | Fat. Grams | Carbohydrate Grams. | Calories. |
|---|---|---|---|---|
| Oatmeal (dry) ... | 5 | 2 | 20 | 110 |
| Meat (uncooked, lean) | 6 | 2 | 0 | 40 |
| Meat (cooked, lean)... | 8 | 3 | 0 | 60 |
| Broth ... ... | 0·7 | 0 | 0 | 3 |
| Potato ... ... | 1 | 0 | 6 | 25 |
| Bacon ... ... | 5 | 15 | 0 | 155 |
| Cream 40% ... | 1 | 12 | 1 | 120 |
| „ 20% ... | 1 | 6 | 1 | 60 |
| Milk ... ... | 1 | 1 | 2 | 20 |
| Bread ... ... | 3 | 0 | 18 | 90 |
| Butter ... ... | 0 | 25 | 0 | 240 |
| Egg (one) ... | 6 | 5 | 0 | 75 |
| Brazil Nuts ... | 5 | 20 | 2 | 210 |
| Orange or Grape Fruit | 0 | 0 | 10 | 40 |
| Vegetables 5 and 10% group... ... | ·05 | 0 | 1 or 2 | 6 or 10 |

It will be seen that these estimates differ slightly from those given by Leyton and by Cammidge. They are rather more generous, in that they give a wider scope with the five and ten per cent. vegetables.



*Grains of sugar in red.*

*Ounces of urine in black*

With the various lists given there is no difficulty in arranging diets for Europeans and non-vegetarian Hindus in the cold weather, or for the greater part of the year. In the rains and hot weather the problem is more difficult, and for the vegetarian Hindu almost impossible. One can do a good deal with the different varieties of sag (spinach) with patal or pulbul, jhingas, cucumbers, bebhute (a kind of bean), moullies and the different varieties of pumpkins. Ghi (clarified butter), furnishes the fat quite easily, cream is generally available to the Marwari, and channa, the casein of milk, will give the proteid required. The various dâls (legumes) are the ordinary sources of proteid food, but they contain a considerable amount of carbohydrate (green peas 15 per cent.), but they have to be relied on in default of anything else.

Indian patients, especially of the uneducated classes, complain bitterly of the want of food. They are accustomed to bulky meals of rice and chapati, and though they may be assured that they are getting the physiological number of calories, that does not satisfy them if the belly is not comfortably distended.

The following are notes of some well marked cases treated by Allen's method of Alimentary Rest. They are of interest from the varied nationalities of the patients and exemplify both the successes and the difficulties of the method in the East. Only cases treated in hospital are reported; it is not practicable to keep accurate records in private cases.

*European Cases.*—I. Mr. A., Factory Manager, aged 59. Admitted 23rd June, 1916. In poor health for a year or so. Shortness of breath, rapid pulse, frequency of micturition, numbness and weakness of legs. Blood pressure 130 m. m. Sugar 1100 grains per diem. Urine 80 ounces.

Under old-fashioned dietetic treatment at his home, combined with a little opium, the sugar diminished to 100 grains *per diem* with the patient grumbling at the restricted diet and at the opium.

On June 23rd, 1916, he was persuaded to enter Hospital for treatment by alimentary rest. Fasted June 25th and 26th. No sugar for nine days, then 40 grains in 24 hours, disappearing after a day's fast.

Discharged on August 4th, 1916, with no sugar.

Previous day's diet :—

6-30 A.M.—Tea, milk 4 oz., two slices of toast and butter, one half-boiled egg.
9 A.M.—Fish 4 oz., dâl 4 oz., cabbage 4 oz.
1 P.M.—Mutton chop 8 oz., boiled potato 4 oz.
4 P.M.—Tea, milk 4 oz., omelette of 2 eggs, one slice toast and butter.
7 P.M.—Mutton chop 4 oz., French beans 4 oz., milk 8 oz., soup 1 cup.

Since his discharge the patient has been living on an ordinary mixed diet, avoiding rice and jam, and there has been no sugar. His urine examined on December 27th, 1916, showed no sugar, although he had been attending Christmas festivities.

From time to time he has reported himself. At no time has there been any sugar, when the urine was examined in the hospital laboratory, though he admitted sugar on one occasion to his own tests, after an indiscreet indulgence at a dinner party. On 22nd June, 1917, his blood sugar was 0·12 % (by Benedict Cammidge).

He last reported himself on July 28th, 1917. There was then no sugar to Benedict test, greenish precipitate, (urates) to Fehling. Weight 175 lbs. Pulse much slower and stronger. Blood pressure 125 m.m. On the day previous to coming he reported himself he had taken the following food:—

*Chota hazri.*—6 A.M. Large cup of tea, milk 3 oz., toast and butter one slice.

*Breakfast.*—9 A.M. Large cup of tea, 1 boiled egg, milk 3 oz., toast and butter one slice.

*Tiffin.*—12 NOON. One poached egg, stewed fowl with spinach (no potatoes or bread), mung dâl, one plateful of soup.

*Tea.*—4 P.M. One cup of tea, milk 3 oz., one slice toast and butter.

*Dinner.*—8 P.M. Mutton soup, one plateful, half of roast fowl, vegetable marrow, 6 oz., stewed peaches (without sugar), custard pudding—one. Whiskey and soda, 6 P.M.

This man has lost some weight and is probably the better for it. He has markedly improved in every respect except for his peripheral neuritis, his knee jerks remain persistently absent, though the heaviness in the legs has improved under ionic treatment with potassium iodide.

The case was not a severe one and has a high carbohydrate tolerance. Yet, in spite of the mildness, the neuritis is marked and the rapidity of the pulse was enough to cause anxiety.

Case II.—Mr. B., aged 53, admitted March 13th, 1917. Factory Manager, living a very hard and strenuous life. History of thirst and frequency of micturition for the last 13 or 14 years. Sugar detected 10 years ago.

Passes urine about 10 times in 24 hours, about 120 to 160 ounces, no pruritus. Carbuncle 3 years ago, knee jerk present but diminished, no ankle clonus. Vision, myopic—2·5. Blood pressure 160. Complains of numbness of the toes for the last few months. Sugar present 15·4 grains per ounce, urine 120 to 190 ounces. Total sugar 2,800 grains per diem. Fasted two days, sugar absent. Reappeared on 13th day after 2 ounces of bread and 2 ounces of potato, but absent again after a restricted diet.

Left Hospital on April 4th, 1917, no sugar, feeling much better. Urine 56 oz.

Previous day's diet:—

6-30 A.M.—Tea 6 oz., egg 1.

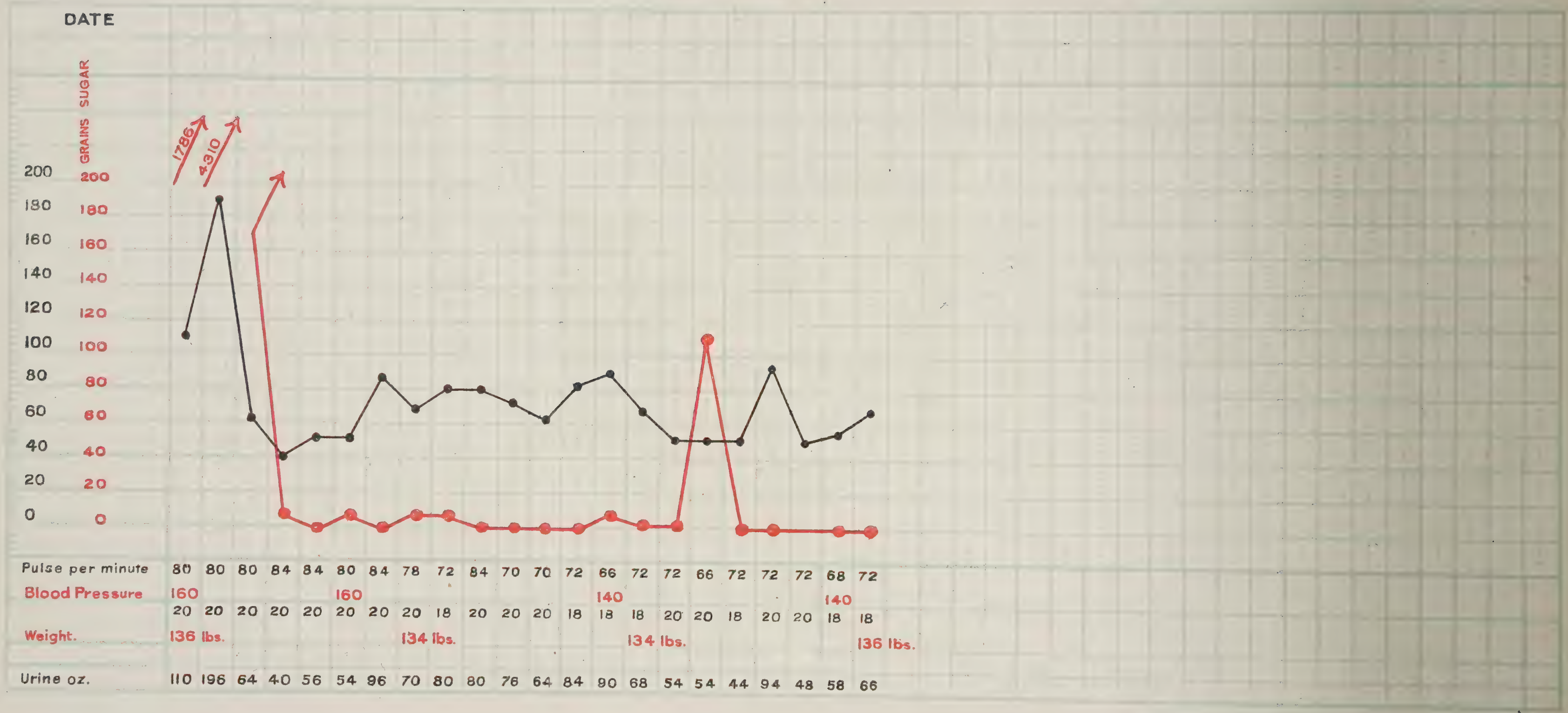
9-30 A.M.—Bacon 2 oz., eggs 2, boiled celery 5 oz., cooked tomato 4 oz.

1-30 P.M.—Mutton 4 oz., cabbage 3 oz., bread ½ oz., butter ½ oz.

4-30 P.M.—Coffee 6 oz., egg 1, butter ½ oz.

7-30 P.M.—Bread ½ oz., cabbage 4 oz., eggs 2, butter ½ oz., cup of soup.

Feeling very well. Gained weight. Is passing a little sugar occasionally, but this is his own fault and it is easily kept in check.



*Grains of sugar in red.*
*Ounces of urine in black*

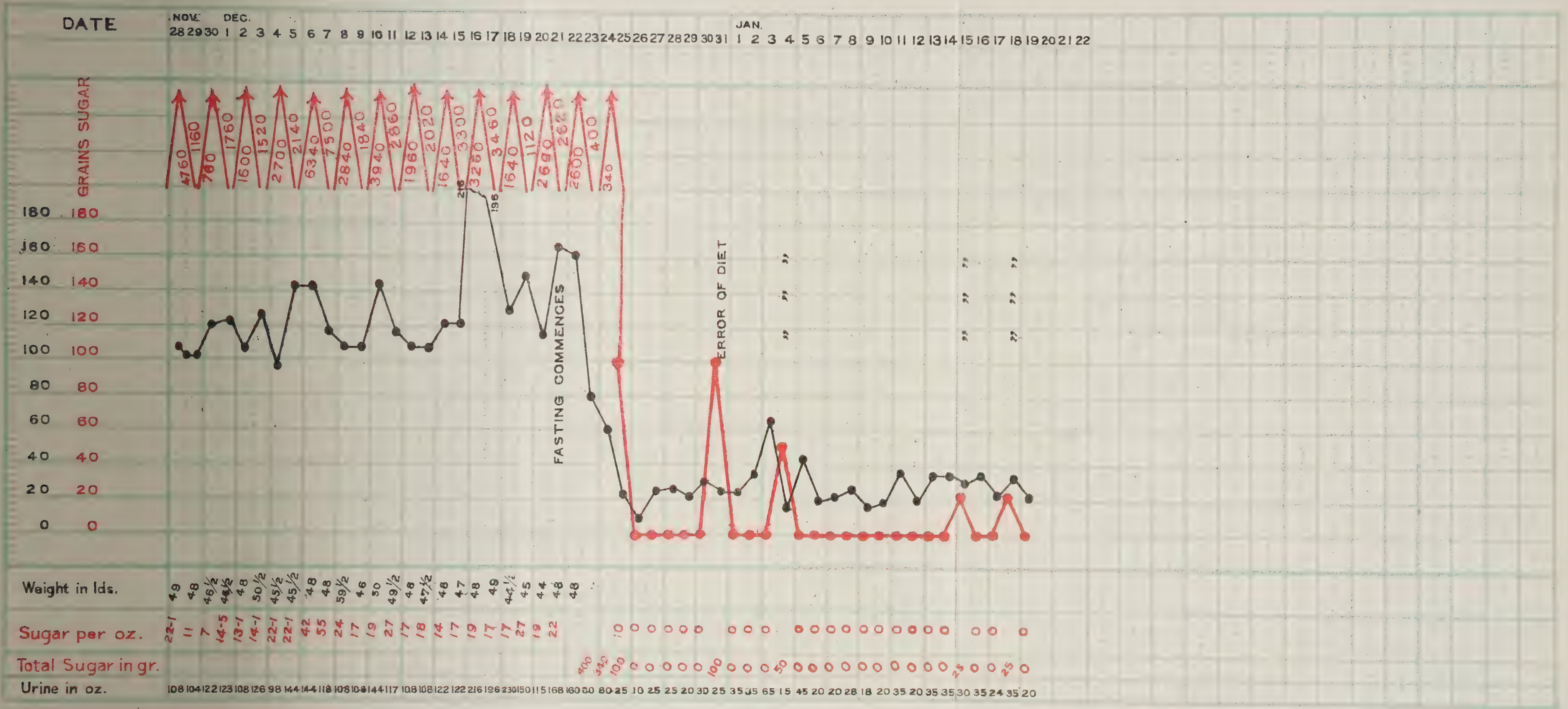
*Indian Cases.*—For obvious reasons these patients are more difficult to manage. The average up-country Hindu of the agricultural class is ignorant and prejudiced on matters of diet and is practically impossible to treat. Cases 3 and 4 are examples of those whom it is almost useless to treat by the Allen method. It was possible to eliminate the sugar temporarily, but not to keep it away permanently.

*Case III.*—B. N., an up-country Hindu (Brahmin), uneducated and dirty, aged 30, admitted for frequency of micturition, voracious appetite and general wasting. Weight 75 lbs. Blood pressure 100 m.m. Sugar, some 4,040 grains daily. A most intractable patient. Would obey no orders. Stole other patients' food even though he was getting three times the full hospital diet. It was impossible at first to get him to fast, so he was given gradually increasing doses of opium, and the food kept as low as possible, though always on full diet. By the time he was taking 20 grains of opium daily, we succeeded in getting the patient to fast for one day, and the sugar came down to 50 grains from the original 4,040. Then he relapsed and spent a day begging in the streets with the expected results. Once more a day's fast got the sugar down to a trace only (Fehling), but a further outbreak made us give up in despair. Even so, the man's weight increased to 80 lbs., his sugar ranged from 400 to 800 grains instead of 4,000, and the patient stoutly declared himself cured!

*Case IV.*—A similar class of case and similarly intractable. Fortunately, at an early stage in the observation, the patient absconded with hospital clothing, was arrested, and was able to continue his cure in jail under stricter supervision.

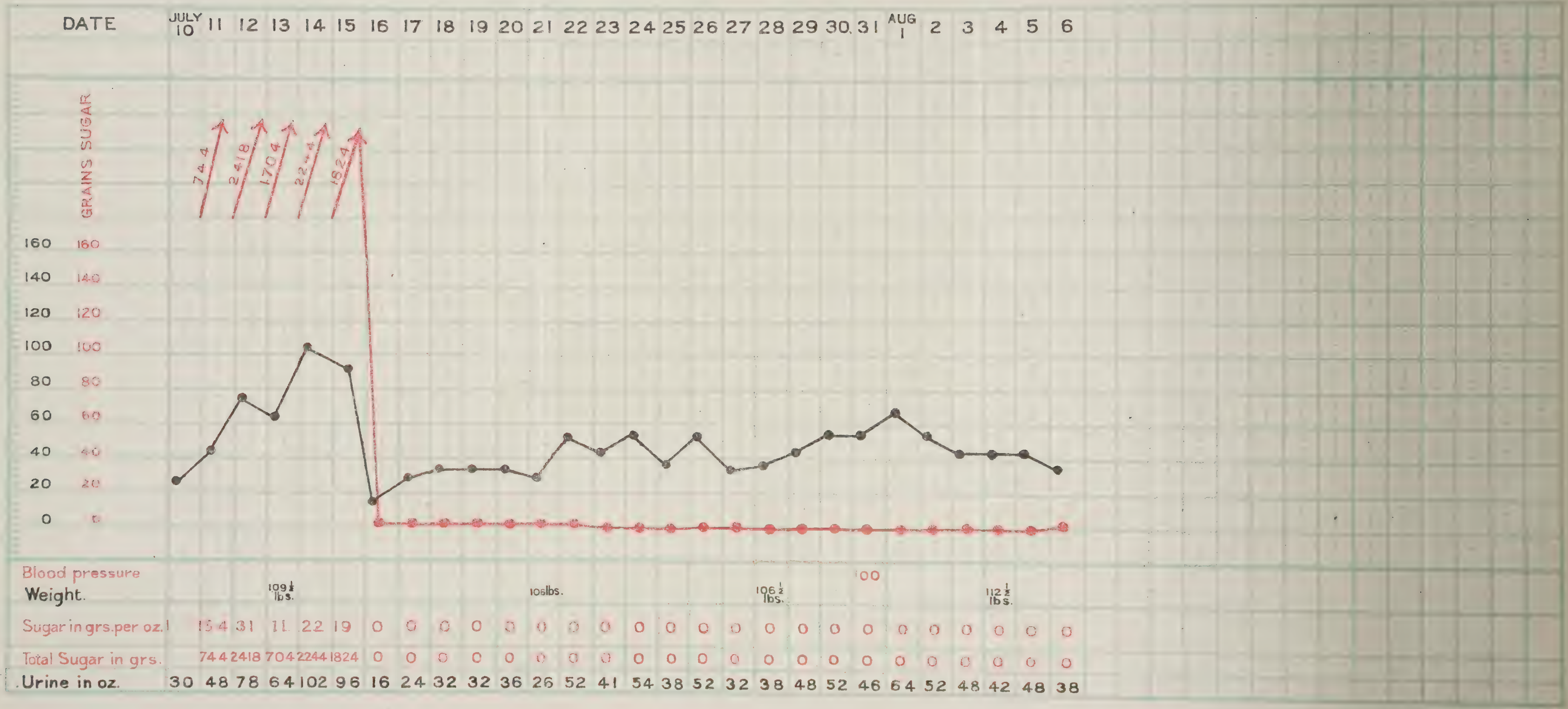
B. T., aged 15 years, an up-country Brahmin beggar, came under observation in November, 1916. Complained of great thirst, frequent micturition, and wasting. Duration 3½ years. Passing over 5,000 grains of sugar daily. Absconded after 36 hours in hospital, stealing hospital property, arrested and remanded to jail. Admitted there on November 26th, 1916. On admission, weight 48 lbs. Urine 200 ounces, sugar 3,560 grains. The urine was always over 100 ounces, and the sugar over 2,000 grains, On one occasion it amounted to 7,840 grains and the next day to 6,365 grains. The weight fluctuated between 45 and 52 lbs. He had an attack of diarrhœa on admission, requiring careful dieting, but after recovery was able to take the usual jail diet of chapati, rice and vegetables. Fasting commenced on December 26th, 1916. He then weighed 45 lbs., had passed 162 ounces of urine and 1,926 grains of sugar in the previous day. After the first day's fast the urine was 81 ounces, sugar 422 grains, weight 45½ lbs. On the next day, urine 62 ounces, sugar 328 grains, weight 43 lbs. On the third day, urine 25 ounces, sugar 55 grains, weight 42 lbs. Fourth day, urine 10 ounces, sugar nil, di-acetic acid present, weight 42½ lbs. Fifth day, urine 24 ounces, sugar nil, weight 42 lbs., di-acetic acid less. Sixth day, urine 24 ounces, di-acetic acid a bare trace, sugar nil, weight 45 lbs.

The boy was depressed at first under treatment, but soon began to feel better and got more cheerful. He was released from jail on January 19th, 1917, weight 45½ lbs. Diet, tea, milk 16 ounces, eggs 2, cabbage ¼, carrot 4 ounces, potato 4 ounces, fish soup 8 ounces, fish 4 ounces, mung dâl 2 ounces, rum 3 ounces, no sugar.



*Grains of sugar in red.*

*Ounces of urine in black*



| DATE | JULY 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood pressure | | | | | | | | | | | | | | |
| Weight. | | | | 109½ lbs. | | | | | | | | 106lbs. | | |
| Sugar in grs. per oz. | | 15.4 | 31 | 11 | 22 | 19 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total Sugar in grs. | | 744 | 2418 | 704 | 2244 | 1824 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Urine in oz. | 30 | 48 | 78 | 64 | 102 | 96 | 16 | 24 | 32 | 32 | 36 | 26 | 52 | 41 |

| DATE | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | AUG 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood pressure | | | | | | | | 100 | | | | | | |
| Weight. | | | | | 106½ lbs. | | | | | | | 112½ lbs. | | |
| Sugar in grs. per oz. | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total Sugar in grs. | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Urine in oz. | 54 | 38 | 52 | 32 | 38 | 48 | 52 | 46 | 64 | 52 | 48 | 42 | 48 | 38 |

*Grains of sugar in red.*

*Ounces of urine in black*

No doubt he would relapse immediately he got back to his old environment, but it was interesting to note how one could reduce the sugar in a young diabetic from 7,000 grains to zero.

*Case V.*—N. D., aged 40, a Brahmin, head constable, came over from Calcutta for treatment. Usual symptoms, frequent micturition, voracious appetite, great thirst and general wasting. Tongue dry. No enlargement of liver and spleen. Bowels regular.

Heart and lungs normal. Blood pressure 100. No dimness of vision, knee jerks present. Weight 109½ lbs. Sugar varied from 1,700 to 2,400 grains daily. Urine 80 to 100 ounces daily. Sugar absent after one day's fast and has remained absent. Weight dropped to 106 lbs. on July 21st, 1917, and has since steadily increased. It is to-day (August 16th, 1917), 120 lbs.

This patient is fairly educated and intelligent, so has been easier to feed; his diet has been practically that of the ordinary Bengali patient. Yesterday he had the following diet, which is estimated to contain 375 grams of carbohydrate, 93 grams of proteid and 75 grams of fat. The calories are 2297.

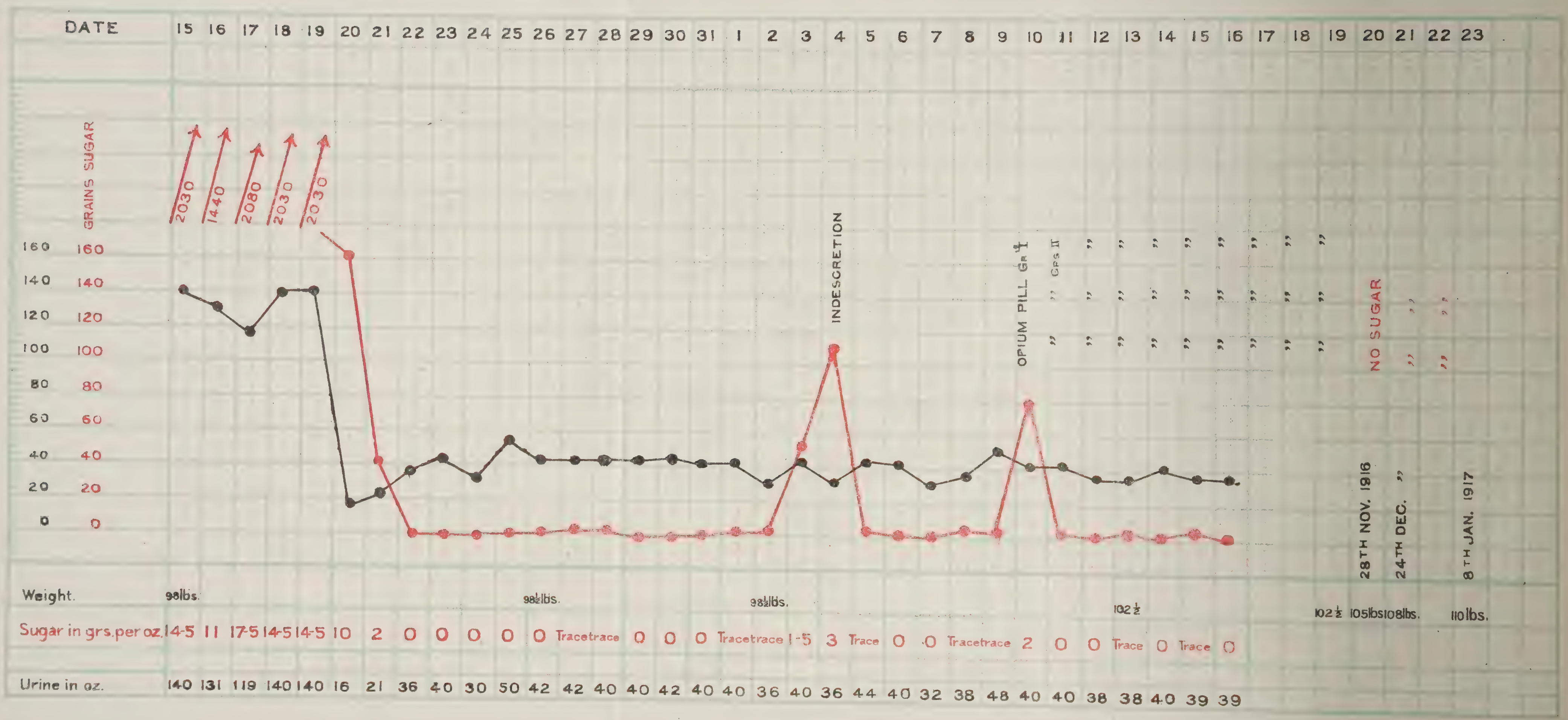
*Diet of* 15*th August* 1917.

| | |
|---|---|
| Green Vegetables ... | 21 ounces |
| Chapati .. ... | 18 ,, |
| Potato ... ... | 1 ounce |
| Dâl ... ... ... | ½ ,, |
| Fish ... ... ... | 5 ounces |
| Soup (fish) ... ... | 7 ,, |
| Fat ... ... ... | 2¼ ,, |
| Milk ... ... ... | 8 ,, |

*Case VI.*—M. L., aged 35, a Marwari cloth merchant, suffered from diabetes for many years and had tried many treatments. First seen on September 7th, 1916, passing 2·5 per cent. of sugar. This was a most difficult case to treat, for he would not come into hospital, and would not have a daily sugar estimation done. Moreover, as a Jain, he was a strict vegetarian, and eggs, fish, and meat were forbidden to him. After two days' fast the sugar disappeared from his urine and did not return whilst under observation. Seen again on September 18th, 1916, no sugar, had gained a little weight. He did not come again till December 29th, 1916. He had then put on a good deal of weight and said he was quite well. He was eating the ordinary Marwari diet of rice, chapati, and sweetmeats, and proposed to continue doing so, but he was passing 2 grains of sugar per ounce. On January 9th, 1917, after two fast days at an interval of a week, and the same diet, he had no sugar.

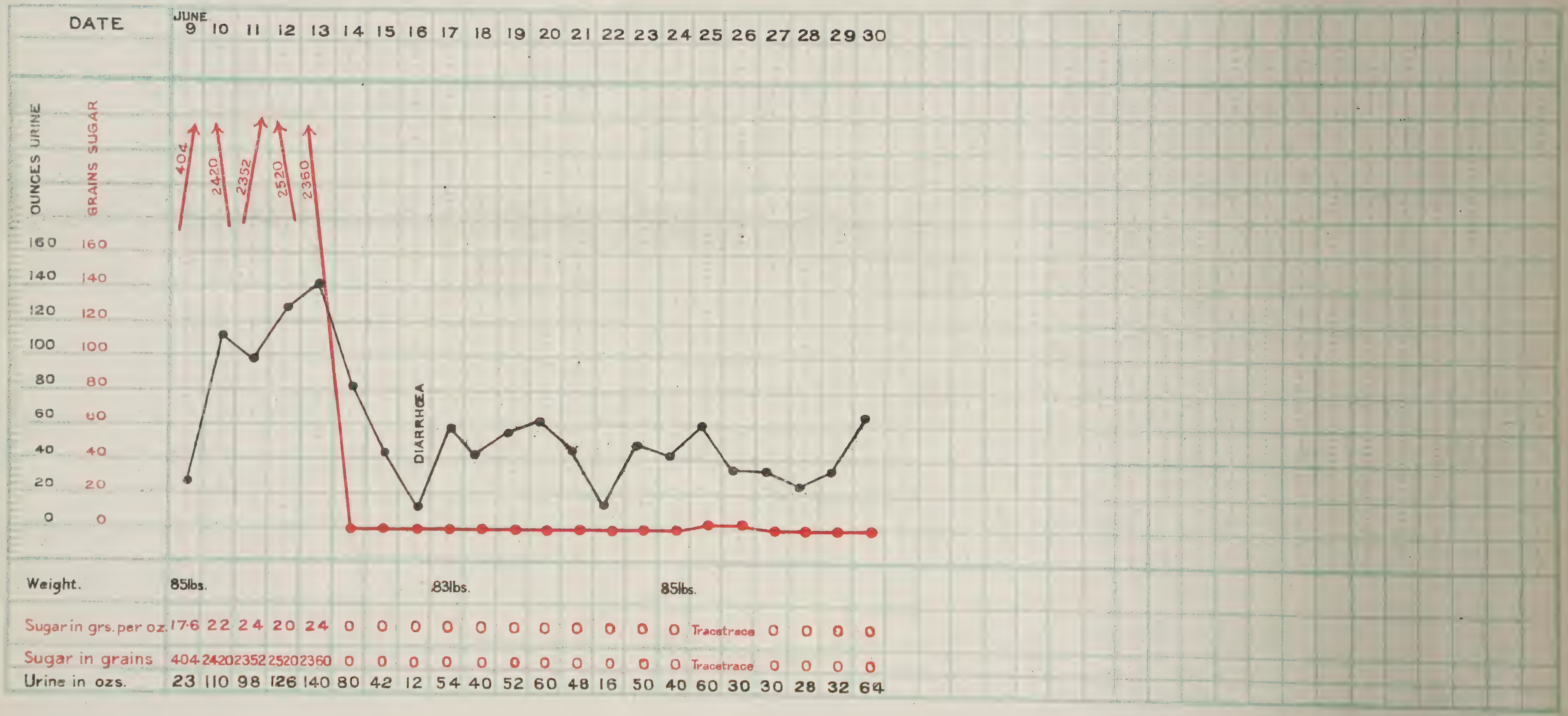
This man has come up once or twice since he has relapsed, will not restrict his diet, and is now passing as much sugar as ever he did.

The diets I employ now for these cases, and for Bengali cases, is detailed below. It is the best one can do, but it is far from perfect; they are too expensive for the ordinary clerk or petty official to adhere to, and suffer from the great defect of not being sufficiently filling. They rule out many of the most favourite and most satisfying articles of diet, such as rice in large quantity, dâl, and milk. Still, with a little perseverance, it can be done, as the following cases show:—



*Grains of sugar in red.*

*Ounces of urine in black.*



| DATE | JUNE 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Weight. | 85lbs. |  |  |  |  |  |  |  | 83lbs. |  |  |  |  |  |  | 85lbs. |  |  |  |  |  |  |
| Sugar in grs. per oz. | 17·6 | 22 | 24 | 20 | 24 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | Trace | trace | 0 | 0 | 0 | 0 |
| Sugar in grains | 404 | 2420 | 2352 | 2520 | 2360 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | Trace | trace | 0 | 0 | 0 | 0 |
| Urine in ozs. | 23 | 110 | 98 | 126 | 140 | 80 | 42 | 12 | 54 | 40 | 52 | 60 | 48 | 16 | 50 | 40 | 60 | 30 | 30 | 28 | 32 | 64 |

*Grains of sugar in red.*

*Ounces of urine in black.*

## BENGALI CASES.

### *Bengali Diet.*

*Case VIII.*—A. T. B., Kayastha, aged 48 years, a clerk in the East Indian Railway. Admitted on August 15th, 1916, for frequency of micturition, great thirst and general weakness. Duration four months; urine shows from 3 to 6 per cent. of sugar. Symptoms and physical signs, weakness of legs, absent knee jerk, unsteady gait, poor vision, burning sensation in hands and feet. Blood pressure 120 m.m. Weight 98 lbs. Takes one grain of opium daily. Passes 148 ounces of urine daily and from 1,700 to 2,000 grains of sugar.

This man had no treatment for the first five days and then starved for three days. The urine diminished to 20 ounces daily, sugar disappeared. Diet gradually increased, a trace of sugar on one or two occasions, probably due to dietary indiscretions. Left hospital on September 20th, 1916. Had taken rice for several days; no sugar in urine. Weight 102½lbs. Came back again after two months, during which he said he had taken ordinary Bengali diet, no sugar. Weight 105 lbs. Seen again on December 24th, 1916, weight 108 lbs., no sugar. Is at work and says he is quite well. On January 1st, 1917, weighs 110 lbs., diet on the previous day was that of a normal Bengali, sweetmeats and rice, no sugar. Is doing his daily work.

*Case IX.*—Jatadhari Kundu, Bengali, weaver by caste, aged 40, admitted June 9th, 1917, from Hughli.

*History, etc.*—Frequent micturition, general wasting, voracious appetite, great thirst, duration nearly 2 years.

*Physical signs.*—Tongue dry and wrinkled. Ravenous appetite. Constipated. Liver slightly enlarged, circulatory and respiratory systems normal, knee jerks very slightly present, giddiness at times, urine varied from one hundred to one hundred and forty ounces daily, the sugar was about 2,400 grains per diem.

After five days on ordinary diet, to find out what amount of sugar he was excreting, fasting was commenced. Sugar was absent at the end of the first 24 hours, and remained absent (except for an unestimable trace in two days) for the remainder of his stay in hospital. The standard diet as arranged for Bengalies was given.

The patient asserted that he was cured, and after 22 days' treatment declined to stay any longer in hospital.

*His diet before leaving was :—*

| | |
|---|---|
| Green Vegetables ... | 20 ounces |
| Chapati ... ... | 16 ,, |
| Potato ... ... | 1 ounce |
| Fish ... ... ... | 6 ounces |
| Eggs ... ... | Four |
| Soup ... ... | 8 ounces |
| Fat ... ... ... | 2¼ ,, |
| Milk ... ... ... | 4 ,, |
| Protein ... ... | 128 grams |
| Fat ... ... ... | 92 ,, |
| Carbohydrate ... ... | 322 ,, |
| Calories ... ... | 2774. |
| Sugar ... ... | *Nil.* |

### Specimen diet for Indian patients.

This diet is most suitable for Bengalies, as it includes fish, eggs and meat. It probably contains too much fat for severe cases, and the caloric value is increased too rapidly. This, of course, must be modified as required. At the time of writing the diet is being given to three cases, a Hindustani Brahmin head constable (case 5), a Bengali Brahmin sub-inspector, and a Bengali B class prisoner in the jail. In the case of the Hindustani, the eggs are omitted, the fish and meat might be cut out, if necessary on religious grounds. The vegetables are those obtainable in the rains in Bengal. In the cold weather—cabbage, cauliflower, etc., might be utilised.

So far as my experience goes, there is no doubt that the Allen Treatment by alimentary rest is the best method for use in the East, certainly for people with any intelligence. The difficulty of treating any chronic medical case in India is well known, for the patient's faith, or that of his friends soon wavers, especially when it is undermined by the ever-present charlatan of the bazaars. But the Allen Method is easily reconciled to traditional Eastern methods. Fasting is enjoined in most Indian religions and is extensively adopted by Kavirajes and Hakims. If the preliminary fasting is timed to coincide with the *Ekadesi* or similar days of religious abstention it will be easier to obtain the patient's acquiescence, or what is more important—the consent of the female members of his family.

Then, too, the great majority of Indian cases are not of the severe variety; the carbohydrate tolerance is relatively high, though this is not always the case.

The difficulty appears to lie in persuading the patient to persist with the treatment, not for a few weeks, but for months or years, and this is particularly the case with imperfectly educated people. The patient (or more particularly the patient's friends) is worried that he cannot take a full meal of rice or chapati, and that the weight is less than it used to be. I see no objection to giving opium, or to allowing the patient to continue his daily opium if he be in the habit of taking it. Opium is useful in that it slows the digestive functions, and possibly gives the organism a little more time to deal with carbohydrate and sugar.

*Fast until sugar free—usually 48 hours.*

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *1st day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar ; eggs 2. | | | | |
| | 10 A.M. | Sag. 2 oz., radish 4 oz. | | | | |
| | 1 P.M. | Mutton tea 1 pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar ... | 29 | 10 | 6 | 246 |
| | | *2nd day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Sag. 4 oz., radish 6 oz., patal 4 oz., cooked in mustard oil 2 oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Eggs 2, tea 1 cup without milk and sugar, patal 4 oz., sag. 4 oz., radish 2 oz., cucumber 4 oz., cooked in ghee 2 oz, spices 2 oz. ... | 52 | 120 | 28 | 1488 |
| | | *3rd day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Sag. 4 oz, cabbage 4 oz., patal 4 oz., radish 4 oz., cooked in ghee 2 oz., spices 2 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 1 P.M. | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, fish 4 oz, patal 4 oz., sag. 4 oz., cabbage 4 oz., radish 4 oz., cooked in mustard oil 2 oz., spices 2 oz. ... ... | 66 | 118 | 32 | 1522 |
| | | *4th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M | Fish 4 oz, sag. 4 oz., patal 4 oz., cucumber 4 oz., cooked in ghee 2 oz., spices 2 oz. | | | | |
| | 1 P.M. | Mutton tea 1 pint. ... | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, fish 4 oz., cucumber 4 oz., patal 4 oz., radish 4 oz., cooked in mustard oil 2 oz, spices 2 oz. ... | 86 | 126 | 24 | 1634 |
| | | *5th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., patal 4 oz., potato 2 oz., cucumber 4 oz., ghee 2 oz., spices 2 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, patal 2 oz , butter 1 oz , mustard oil 2 oz , spices 2 oz ... | 62 | 157 | 22 | 1860 |
| | | *6th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., potato 2 oz., cucumber 4 oz., patal 4 oz., ghee 2 oz., spices 2 oz. | | | | |
| | 1 P.M. | Mutton soup ½ pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, patal 6 oz., cucumber 6 oz., mustard oil 2 oz., butter 1 oz., spices 2 oz. ... ... | 79 | 157 | 32 | 1950 |
| | | *7th day after sugar free.* | | | | |
| | | Fast day. | | | | |
| | | *8th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 3 oz , ghee ¾ oz., sag. 2 oz., patal 3 oz., cucumber 3 oz., spices 2 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, ghee ¼ oz., patal 3 oz., cucumber 3 oz., fish 3 oz, spices 2 oz., mustard oil 2 oz | 67 | 107 | 14 | 1324 |
| | | *9th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., potato 2 oz., cucumber 4 oz., patal 4 oz., radish 2 oz., mustard oil 2 oz, spices 2 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, patal 4 oz., sag. 4 oz., cucumber 4 oz., ghee 2 oz, spices 2 oz. ... | 78·6 | 132 | 34 | 1716 |
| | | *10th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., potato 2 oz., brinjal 2 oz., cucumber 4 oz., patal 4 oz., mustard oil 2 oz., spices 2 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, butter 1 oz., patal 4 oz., sag. 4 oz., brinjal 4 oz., mustard oil 2 oz., spices 2 oz. ... | 78·6 | 157 | 34 | 1956 |
| | | *11th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2 | | | | |
| | 10 A.M. | Potato 2½ oz., cucumber 4 oz., patal 4 oz, sag. 6 oz., fish 6 oz., mustard oil 2 oz., spices 2 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, butter 1 oz, patal 4 oz., brinjal 4 oz., cucumber 4 oz., mustard oil 2 oz., spices 2 oz. ... | 81·1 | 157 | 41 | 1995 |
| | | *12th day after sugar free* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., potato 3 oz., patal 4 oz., brinjal 4 oz., cucumber 4 oz., sag. 4 oz., mustard oil 2 oz., spices 2 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, butter 1 oz., patal 4 oz., brinjal 4 oz., cucumber 4 oz., mustard oil 2 oz., spices 2 oz. ... | 81·6 | 157 | 44 | 2010 |
| | | *13th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., potato 4 oz., patal 4 oz., cucumber 6 oz, brinjal 4 oz., mustard oil 2 oz., spices 2 oz. | | | | |
| | 1 P M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, butter 1 oz., patal 4 oz., brinjal 4 oz., cucumber 4 oz., mustard oil 2 oz., spices 2 oz. ... | 82·6 | 157 | 50 | 2040 |
| | | *14th day after sugar free.* | | | | |
| | 8 A.M | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Chapati 3 oz., potato 1 oz., patal 4 oz., brinjal 4 oz., fish 6 oz., mustard oil 2 oz., spices 2 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 1 P.M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, butter 1 oz., patal 4 oz., brinjal 4 oz., cucumber 4 oz., mustard oil 2 oz., spices 2 oz. ... | 85·6 | 157 | 80 | 2184 |
| | | *15th day after sugar free.* | | | | |
| | | Fast-day. | | | | |
| | | *16th day after sugar free.* | | | | |
| | | Repeat diet of 14th day | 85·6 | 157 | 80 | 2184 |
| | | *17th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Chapati 3 oz., potato 2 oz., patal 4 oz., brinjal 4 oz., fish 6 oz., mustard oil 1 oz., spices 1 oz | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 3 oz., patal 4 oz., brinjal 4 oz., cucumber 4 oz., mustard oil 1 oz., spices 1 oz. | 95·6 | 82 | 140 | 1764 |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *18th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Chapati 6 oz., with ghee ¼ oz., potato 2 oz., patal 4 oz, cucumber 4 oz., fish 6 oz., mustard oil 1 oz., spices 1 oz | | | | |
| | 1 P.M. | Fish soup 8 oz | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 3 oz., patal 4 oz., brinjal 4 oz, cucumber 4 oz., mustard oil 1 oz., spices 1 oz. ... ... | 104·6 | 88·25 | 194 | 2094 |
| | | *19th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A M. | Chapati 6 oz, with ghee 1 oz., potato 1 oz., fish 6 oz, patal 4 oz., karala 2 oz., radish 2 oz., brinjal 4 oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 6 oz., patal 4 oz, cucumber 4 oz, sag 4 oz, mustard 1 oz., spices 1 oz. ... | 114·6 | 107 | 246 | 2538 |
| | | *20th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., chapati 7 oz., patal 4 oz., cabbage 4 oz, dharos 4 oz, potato 1 oz., ghee ¼ oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 7 oz., patal 4 oz., brinjal 4 oz, mustard oil 1 oz, spices 1 oz, ghee ¼ oz. ... | 118·6 | 94·5 | 278 | 2574 |
| | | *21st day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 10 A.M. | Fish 6 oz., chapati 8 oz., patal 4 oz., cabbage 4 oz., dharos 4 oz., potato 1 oz, ghee ¼ oz., mustard oil 1 oz, spices 1 oz. | | | | |
| | 1 P.M | Fish soup 8 oz | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 8 oz, patal 4 oz., brinjal 4 oz., mustard oil 1 oz., spices 1 oz. ... | 124·6 | 88·25 | 314 | 2694 |
| | | *22nd day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz., chapati 8 oz, patal 4 oz., cabbage 4 oz., dharos 4 oz, potato 1 oz., ghee ¼ oz., mustard oil 1 oz, spices 1 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 8 oz, patal 4 oz., brinjal 4 oz., mustard oil 1 oz., spices 1 oz., milk 4 oz. ... ... | 128·6 | 92·25 | 322 | 2774 |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *23rd day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 6 oz, chapati 8 oz., patal 4 oz, dharos 4 oz., potato 1 oz., ghee ¼ oz., mustard oil 1 oz., spices 1 oz., milk 4 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, chapati 8 oz., patal 4 oz., brinjal 4 oz. mustard oil 1 oz. spices 1 oz., milk 4 oz., eggs 2. ... | 130·6 | 96·25 | 326 | 2830 |
| | | *24th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2, milk 4 oz. | | | | |
| | | Fish 3 oz., chapati 4 oz, patal 2 oz., dharos 2 oz., potato ½ oz, ghee ¼ oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P.M. | Fish soup 8 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, chapati 4 oz., patal 2 oz, brinjal 2 oz., mustard oil 1 oz., spices 1 oz., milk 4 oz. ... | 72·1 | 80·25 | 171 | 1777 |
| | | *25th day after sugar free.* | | | | |
| | | Repeated diet of 23rd day ... | 130·6 | 96·25 | 326 | 2830 |
| | | *26th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 5 oz.. chapati 9 oz, brinjal 4 oz., sag 4 oz., potato 1 oz., patal 4 oz., ghee ¼ oz, mustard oil 1 oz., spices 1 oz., moong dál ½ oz, milk 4 oz. | | | | |
| | 1 P.M. | Fish soup 7 oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar. chapati 9 oz., patal 4 oz, brinjal 5 oz., mustard oil 1 oz., spices 1 oz., milk 4 oz. ... ... | 122·59 | 84·30 | 375·25 | 2897·5 |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *27th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2 | | | | |
| | 10 A.M. | Fish 4½ oz., chapati 10 oz., patal 4 oz., brinjal 4 oz., sag. 4 oz., potato 1 oz., ghee ¼ oz, mustard oil 1 oz, spices 1 oz, dâl 1 oz., milk 4½ oz. | | | | |
| | 1 P.M. | Fish soup 6½ oz. | | | | |
| | 6 P.M. | Tea 1 cup without milk and sugar, eggs 2, chapati 10 oz., patal 4 oz., sag 5 oz., mustard oil 1 oz., spices 1 oz., milk 4½ oz. ... | 141·65 | 95·25 | 421·5 | 3276·5 |
| | | *28th day after sugar free.* | | | | |
| | 8 A.M. | Tea 1 cup without milk and sugar, eggs 2. | | | | |
| | 10 A.M. | Fish 4 oz, chapati 9 oz., patal 4 oz., brinjal 4 oz., radish 4 oz., potato 1 oz., ghee ¼ oz., mustard oil 1 oz., spices 1 oz., rice uncooked 1 oz., dâl 1 oz. | | | | |
| | 1 P.M. | Fish soup 6 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk and sugar, chapati 9 oz., patal 4 oz., cucumber 4 oz., mustard oil 1 oz., spices 1 oz., milk 4½ oz., eggs 2 ... | 129·2 | 89·30 | 377·5 | 2991 |

The diet given below is a more gradual one and contains less fat. The increase in caloric value takes place much later on than in the first diet given. It may be used in the more severe cases.—

*Fast until sugar free.*

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *1st day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar. | | | | |
| | 10 A.M. | Sag 1 oz., brinjal 1 oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint (from 1 lb. of mutton) | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, rum 3 oz. in 24 hours ... | 15 | ... | 2 | 72 |
| | | | Rum 3 oz. | | ... | 315 |
| | | | | | | 387 |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *2nd day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar. | | | | |
| | 10 A.M. | Sag. 2 oz., radish 2 oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, sag 1 oz., rum 3 oz. ... | 16 | ... | 5 | 90 |
| | | | Rum 3 oz. | | ... | 315 |
| | | | | | | 405 |
| | | *3rd day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar. | | | | |
| | 10 A.M. | Sag. 2 oz., radish 2 oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Sag. 1 oz., radish 1 oz., tea 1 cup without milk or sugar, rum 3 oz. ... ... | 17 | ... | 6 | 96 |
| | | | Rum 3 oz. | | .. | 315 |
| | | | | | | 411 |
| | | *4th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar. | | | | |
| | 10 A.M. | Sag. 3 oz., radish 2 oz., mustard oil ¼ oz., spices ¼ oz. | | | | |

| Date. | Hours | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 1 P.M. | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, sag. 2 oz, radish 2 oz., rum 3 oz. ... | 18·5 | 7 | 9 | 173 |
| | | | Rum | 3 oz. | ... | 315 |
| | | | | | | 488 |
| | | *5th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar. | | | | |
| | 10 A.M. | Sag. 3 oz., cucumber 3 oz., radish 4 oz., mustard oil ¼ oz., spices ¼ oz | | | | |
| | 1 P.M | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, cucumber, 2 oz. sag. 2 oz., rum 3 oz. ... | 21·5 | 7 | 13 | 197 |
| | | | Rum | 3 oz. | ... | 315 |
| | | | | | | 512 |
| | | *6th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A.M. | Sag. 3 oz., cucumber 3 oz., radish 4 oz., mustard oil ¼ oz., spices ¼ oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint. | | | | |

| Date. | Hours. | Diets. | Pr | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk or sugar, cucumber 2 oz., sag 2 oz, radish 1 oz., rum 3 oz | 27 5 | 11 | 15 | 210 |
| | | | Rum 3 oz. | | .. | 315 |
| | | | | | | 525 |
| | | *7th day after free from sugar.* | | | | |
| | | Day of restricted diet. (*Half ration day*) ... | 13 | 5·5 | 7·5 | 105 |
| | | | Rum 3 oz. | | ... | 315 |
| | | | | | | 420 |
| | | *8th day after free from sugar.* | | | | |
| | | Repeat Diet of 6th day | | | | |
| | | *9th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A.M. | Sag. 3 oz. cucumber 3 oz., radish 4 oz., fish 1 oz, mustard oil ¼ oz, spices ¼ oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, sag 3 oz., patal 3 oz., radish 2 oz., mustard oil ¼ oz, spices ¼ oz., egg 1, rum 3 oz. in 24 hours ... | 26 | 25 | 18 | 450 |
| | | | Rum 3 oz. | | ... | 315 |
| | | | | | | 765 |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *10th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, eggs 2. | | | | |
| | 10 A.M. | Sag. 3 oz., cucumber 3 oz., radish 4 oz., fish 2 oz., mustard oil ½ oz., spices ½ oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint, egg 1. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, sag. 3 oz., patal 4 ozs., pumpkin 3 oz., mustard oil ½ oz., spices ½ oz., rum 2 oz., in 24 hours ... ... | 58 | 46 | 20 | 765 |
| | | | Rum 2 oz. | | ... | 200 |
| | | | | | | 965 |
| | | *11th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, eggs 2. | | | | |
| | 10 A.M. | Sag. 4 oz., patal 4 oz., radish 4 oz., fish 2 oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint, egg 1. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk or sugar, radish 4 oz., pumpkin 2 oz., cucumber 4 oz., mustard oil 1 oz., spices 1 oz., rum 1½ oz., in 24 hours ... | 59 | 71 | 22 | 1017 |
| | | | Rum 1½ oz. ... | | | 150 |
| | | | | | | 1167 |
| | | *12th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, eggs 2 | | | | |
| | 10 A.M. | Sag. 4 oz., patal 2 oz., radish 4 oz., fish 4 oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P.M. | Mutton soup 1 pint, egg 1. | | | | |
| | 6 P.M. | Radish 4 oz., cucumber 4 oz., pumpkin 2 oz., mustard oil 1 oz., spices 1 oz., egg 1, rum 1 oz., in 24 hours ... ... | 70 | 84 | 20 | 1140 |
| | | | Rum 1 oz. .. | | | 100 |
| | | | | | | 1240 |
| | | *13th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, eggs 2. | | | | |
| | 10 A.M. | Sag. 2 oz., potato 1 oz., radish 4 oz., fish 4 oz., mustard oil 1 oz., spices 1 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 1 P.M. | Mutton soup 1 pint, egg 1. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, cucumber 3 oz., radish 4 oz., patal 4 oz., mustard oil 1 oz., spices 1 oz., egg 1, rum 1 oz., in 24 hours ... ... | 69·5<br>Rum 1 oz. | 84 | 23<br>... | 1152<br>100<br>1252 |
| | | *14th day after free from sugar.* | | | | |
| | | Half ration day. | 34¾<br>Rum ½ oz. | 42 | 11·5<br>... | 576<br>50<br>626 |
| | | *15th day after free from sugar* | | | | |
| | | Repeat diet on 13th day. | | | | |
| | | *16th day after free from sugar.* | | | | |
| | 6 A. M. | Tea 1 cup without milk or sugar, eggs 2. | | | | |
| | 10 A. M. | Sag. 2 oz., potato 1 oz., radish 4 oz., fish 4 oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P. M. | Potato 2 oz., eggs 2. | | | | |

| Date. | Hours. | Diets. | Pr | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P M. | Tea 1 cup without milk or sugar, cucumber 3 oz., radish 4 oz., patal 4 oz., mustard oil 1 oz., spices 1 oz., rum 1 oz., in 24 hours ... | 57 | 84 | 35 | 1152 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 1252 |
| | | *17th day after free from sugar.* | | | | |
| | 6 A. M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A. M. | Fish 2 oz., patal 4 oz., radish 4 oz., potato 2 oz., mustard oil 1 oz., spices 1 oz. | | | | |
| | 1 P M. | Potato 2 oz., eggs 2. | | | | |
| | 6 P. M. | Tea 1 cup without milk or sugar, egg 1, fish 2 oz., sag, 4 oz., potato 2 oz., radish 2 oz., mustard oil 1 oz., spices 1 oz., rum 1 oz., in 24 hours ... ... | 61 | 78 | 50 | 1204 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 1304 |
| | | *18th day after free from sugar.* | | | | |
| | 6 A. M. | Tea 1 cup without milk or sugar, egg 1. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 10 A. M. | Fish 2 oz., patal 4 oz., radish 3 oz., sag. 2 oz., mustard oil 1 oz., spices 1 oz, chapati 2 oz., ghee ¼ oz. | | | | |
| | 1 P. M. | Potato 2 oz., eggs 2. | | | | |
| | 6 P. M. | Tea 1 cup without milk or sugar, egg 1, fish 2 oz, potato 2 cz., cucumber 2 oz., radish 4 oz, mustard oil 1 oz., spices 1 oz., rum 1 oz., in 24 hours. | 65·5 | 84 | 75 | 1390 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 1490 |
| | | *19th day after free from sugar.* | | | | |
| | | Repeat diet of 18th day, but add 2 oz. chapati in the evening ... ... | 71·5 | 84 | 111 | 1570 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 1670 |
| | | *20th day after free from sugar.* | | | | |
| | 6 A. M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A. M. | Fish 2 oz, potato 2 oz., cucumber 2 oz., radish 2 oz, sag. 2 oz., mustard oil 1 oz, spices 1 oz, chapati 3 oz., ghee ¼ oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 1 P. M | Potato 2 oz., eggs 2. | | | | |
| | 6 P . | Tea 1 cup without milk or sugar, egg 1, fish 2 oz., potato 2 oz, cucumber 2 oz., radish 3 oz., mustard oil 1 oz.. spices 1 oz.. chapati 3 oz., rum 1 oz., in 24 hours ... | 77·5 | 84 | 119 | 1786 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 1886 |
| | | *21st day after free from sugar.* | | | | |
| | | Half ration day .. | 38·7 | 42 | 59·5 | 893 |
| | | | Rum ½ oz. | | ... | 50 |
| | | | | | | 943 |
| | | *22nd day after free from sugar.* | | | | |
| | | Repeat diet of 20th day. | | | | |
| | | *24th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A.M. | Fish 2 oz.. potato 2 oz., patal 2 oz.. radish 2 oz., sag. 2 oz., mustard oil 1 oz., spices 1 oz., chapati 4 oz., ghee ¼ oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 1 P.M. | Potato 2 oz , eggs 2. | | | | |
| | 6 P M. | Tea 1 cup without milk or sugar, egg 1, fish 2 oz., potato 2 oz., cucumber 2 oz., pumpkin 3 oz., mustard oil 1 oz., spices 1 oz., chapati 4 oz., ghee ¼ oz., rum 1 oz , in 24 hours ... | 83·5 | 90·5 | 194 | 2026 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 2126 |
| | | *24th day after free from sugar.* | | | | |
| | | Repeat diet of 23rd day, but add 1 oz, chapati in the morning and 1 oz. in the evening ... | 89·5 | 90·5 | 230 | 2206 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 2306 |
| | | *25th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A.M. | Fish 2 oz., potato 2 oz., brinjal 2 oz., sag. 2 oz , radish 2 oz., mustard oil 1 oz., spices 1 oz , chapati 6 oz , ghee ¼ oz. | | | | |
| | 1 P.M. | Eggs 2, potato 2 oz. | | | | |

| Date. | Hours. | Diets. | Pr. | Fat | CH. | Calories. |
|---|---|---|---|---|---|---|
| | 6 P.M. | Tea 1 cup without milk or sugar, egg 1, fish 2 oz., potato 2 oz., patal 2 oz., brinjal 2 oz., cucumber 2 oz., mustard oil 1 oz., spices 1 oz., chapati 6 oz., ghee ¼ oz., rum 1 oz., in 24 hours .. | 96 | 90·5 | 267 | 2392 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 2492 |
| | | *26th day after free from sugar.* | | | | |
| | 6 A.M. | Tea 1 cup without milk or sugar, egg 1. | | | | |
| | 10 A.M. | Fish 2 oz., potato 2 oz., brinjal 2 oz., sag 2 oz., radish 2 oz., mustard oil 1 oz., spices 1 oz., chapati 6 oz., ghee ¼ oz., milk 2 oz. | | | | |
| | 1 P.M. | Potato 2 oz., eggs 2. | | | | |
| | 6 P.M. | Tea 1 cup without milk or sugar, fish 2 oz., egg 1, potato 2 oz., patal 2 oz., brinjal 2 oz., cucumber 2 oz., mustard oil 1 oz., spices 1 oz., chapati 6 oz., ghee ¼ oz., rum 1 oz., in 24 hours ... | 98 | 92·5 | 278 | 2432 |
| | | | Rum 1 oz. | | ... | 100 |
| | | | | | | 2532 |

| Date | Hours. | Diets. | Pr. | Fat. | CH. | Calories. |
|---|---|---|---|---|---|---|
| | | *27th day after free from sugar.* | | | | |
| | | Repeat diet of 26th day, but add 2 oz. of milk in the evening ... | 100 | 94·5 | 275 | 2472 |
| | | | Rum 1 oz. ... | | | 100 |
| | | | | | | 2572 |
| | | *28th day after free from sugar.* | | | | |
| | | Half ration day ... | 50 | 47·2 | 137·5 | 1236 |
| | | | Rum ¼ oz. ... | | | 50 |
| | | | | | | 1286 |

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# CHAPTER V.

## TREATMENT.—(*Continued.*)

WHILE the value of the Allen Treatment is being increasingly appreciated, it has not yet received world-wide adoption, and it is therefore necessary to detail some of the older methods.

All responsible observers unite in agreeing that partial or modified fasting treatments are not to be recommended. The Allen Method should be strictly carried out. But certain modified and special diets have been published and attention must be given to them.

Williamson (*Lancet*, 28th April, 1917) gives the following:—

"A routine examination of the patient is first made, complications noted, and the abnormalities of the urine determined. When certain complications (affections of the lungs, heart, and kidneys, etc.) are detected, a special form of treatment may be required and a rigid diabetic diet may not be desirable. Such cases will not be considered in this article.

When the case is one of diabetes without these complications, a diabetic diet will usually be suitable.

When the case is one of diabetes without these complications, a diabetic diet will be usually suitable; but the form of the disease must first be determined. When the urine gives persistently a claret colouration with perchloride of iron, and if this is not due to drugs, and if other indications of decided acidosis can be detected, the case requires very careful and special treatment; these cases will be briefly considered at the end of this article.

If, then, no complications can be detected which require a special form of treatment, and if the urine should give no reaction with perchloride of iron, and no other signs of decided acidosis can be detected, the following course of treatment may then be carried out.

### *Course of Treatment.*

I. The patient is first placed on Diet I—an ordinary rigid diabetic diet, but with the addition of 3 oz. of white bread *daily*.

II. If the glycosuria is not checked in a short time (few days), the white bread is promptly replaced by a pure diabetic bread (free from starch, or only containing the smallest amount thereof), such as biogene bread and ponos biscuits, protein bread, etc.—Diet II.

III. If this fails after a fair trial of 10 or 14 days, the following Diet III is often successful if the patient is able to rest at home on a sofa for a short time.

For *one week* only food is taken in small quantities every 2 hours from 8 A.M. to 10 P.M., and the foods are eggs, milk, cream, custard, coffee, tea, and beef-tea. The following is the diet sheet :—

8 A.M.—Tea or coffee with one tablespoonful of cream. One egg (poached, buttered, or boiled).

10 A.M.—One glass of warm milk (10 oz.).

12 noon.—Custard unsweetened (containing one egg and half-a-pint of milk).

2 P.M.—One glass of warm milk (10 oz.) or cream (1 oz.) in warm beef-tea (10 oz.).

4 P.M.—Tea with one tablespoonful of cream ; one egg (poached, buttered, or boiled).

6 P.M.—Cream (1 oz.) in 10 oz. of warm beef-tea.

8 P.M.—One glass of warm milk (10 oz.).

10 P.M.—Cream (1 oz.) in warm beef-tea (10 oz.).

In this diet (or in the modification following) the patient receives daily 3 or 4 eggs, 2 to 3 oz. of cream, 2 pints of milk, in addition to tea, coffee, and beef-tea.

The various small meals may be changed in order or altered if the patient desires. Thus, at 2 P.M. cream and beef-tea may be taken in place of milk, and a glass of milk at 6 P.M. in place of cream and beef-tea. At 8 P.M. one egg beaten up and added to 10 oz. of warm beef-tea may be taken in place of the glass of milk, or a poached egg and half a pint of beef-tea if the patient prefers. At 10 P.M. a glass of milk may be taken in place of cream and beef-tea. Also if the patient prefer an egg (poached or boiled) and a glass of milk may be taken at 12 in place of custard.

This diet often checks the glycosuria in a few days or by the end of the week. If the glycosuria is not checked by the end of the week, the diet may be continued for a few days longer; but if it then fails it should be discontinued and the patient placed back on Diet II for a few days. If the Diet III has failed to check the glycosuria, the patient may be placed on Diet IV of casein or biogene and cream.

IV. Diet IV of casein, or biogene and cream.

The patient is given for seven or ten days a glass of casein, or biogene and cream with water every two hours from 8 A.M. to 10 P.M. The casein preparation lait-proto No. 6 will probably be the most satisfactory for the majority of patients. A cup of tea or beef-tea, or both, may be also taken, if the patient desires, twice a day.

The mixture of casein and cream and water is prepared as follows: One tablespoonful of casein, or lait-proto No. 6, one tablespoonful of cream; mix well in a tumbler with a spoon, then add hot water (or cold if preferred) very gradually, mixing well until the tumbler is full. (The fluid may be sweetened with saccharin, or flavoured with nutmeg if desired.)

In place of *one* tablespoonful of casein *two* tablespoonfuls of biogene may be used if the patient does not like the taste of the casein. It should be mixed with *two* tablespoonfuls of cream and the water added as just described.

Many patients take the casein or biogene quite well, others find both unpalatable, and some cannot take either. Lait-Proto No. 6 may be taken quite well when ordinary casein or biogene cannot be taken.

If we wish to avoid using diabetic breads instead of ollowing the order just indicated the trial of Diet II may be omitted, and if Diet I fails Diet III may be tried at once; or if the patient likes the taste of casein or biogene, and can be seen daily, Diet IV may be tried before Diet III (at once after Diet I).

I have found Diet III (eggs, cream, milk, beef-tea, etc., as indicated) of very great service; it is simple, palatable, and often very successful. It can be taken by nearly all patients and it is less expensive than diabetic food. It can be usually taken quite well by patients who cannot take casein or biogene. It is very useful in the case of diabetic children. It is often very successful both in mild cases and cases of medium severity, and sometimes in severe cases. In some cases, like all other treatment, it fails. It often removes the glycosuria promptly in a few days or a week, though an ordinary rigid diabetic diet had previously failed. After taking this diet for seven or ten days often the patient may then take ordinary rigid diet for a long period without glycosuria returning, though previously on such a diet the glycosuria had continued. Probably it is not so often successful as the treatment with casein or biogene, but if it fails the latter treatment may then be tried, or a combination of the two methods may be tried. Diet III or IV should be continued for seven or ten days only. They are to be regarded as methods of dietetic treatment which are usually much more successful than an ordinary rigid diabetic diet in removing the glycosuria temporarily, and the after-effects probably continue longer. If later the sugar returns in the urine the methods may be repeated. Diet IV may be successful when Diets II and III have failed. In a few severe cases with persistent diacetic acid reaction in the urine Diet III or IV removes the diacetic acid as well as the sugar, but they are not always suitable in such cases.

After Diet III or IV has been tried for seven or ten days the patient is placed back on Diet II if the glycosuria continues. If the glycosuria has been checked, then the Diet III or IV is gradually changed. One ponos biscuit is allowed at 8 A.M. and 4 P.M. with the egg and tea. One or two days later bacon and green vegetables and custard are allowed at 1, in place of the meals at 12 and 2, and later Diet V and then Diet II are allowed.

Of course, the Diets III and IV will not be successful in all cases, but they are always worthy of a careful trial, except when the urine persistently gives a marked claret colouration with perchloride of iron. In these cases caution is necessary, and sometimes Diets III and IV are not advisable.

V. If all the diets already described have failed to check the glycosuria, then for a short time a diet of suitable vegetables, eggs, and fatty foods may be tried (Diet V).

*Breakfast:* Coffee (or tea) with cream; eggs (buttered, poached, or boiled), or tomatoes or mushrooms; suitable diabetic bread or biscuits; butter. *Dinner:* Bacon cooked in any way. Any of the following vegetables: cabbage, cauliflower, Brussels sprouts, turnips, French beans, spinach, broccoli, boiled walnuts, asparagus, vegetable marrow, tomatoes, mushrooms, salad, lettuce, cucumber, watercress, celery, radishes. Custard: Suitable diabetic biscuits or bread. *Tea or evening meal:* Tea and cream; eggs (buttered, poached, or boiled); or Welsh rarebit, cheese; or salad, lettuce, cucumber, mushrooms, tomatoes, boiled egg and spinach, watercress; suitable diabetic bread; butter.

Or the following diet may be tried for a short time:—

*Breakfast:* Tea or coffee with cream; one egg and tomatoes or mushrooms or spinach; also one ponos biscuit or other similar biscuit. *At* 11 *a.m.:* Two tablespoonfuls of cream in half a pint of warm beef-tea. *Dinner* (12-30 *or* 1): Beef or tomato soup or one sardine and one ponos biscuit. Afterward one or more of the following vegetables: Cabbage, cauliflower, Brussels sprouts, vegetable marrow, broccoli (curly green), boiled celery, asparagus, French beans, turnips, tomatoes, mushrooms, boiled walnuts, Brazil nuts sliced and fried in butter. Custard; stewed rhubarb, gooseberries, or *cranberries* with cream. *Tea* (4 *to* 5): Tea with cream, tomatoes or asparagus or celery or mushrooms, lettuce, salad, cucumber, jelly; one ponos biscuit. *Supper:* Beef-tea and cream (as at 11).

VI. If these diets fail to check the glycosuria then drugs may be tried. The drugs which I have found most useful are: sodium salicylate, aspirin, and salicylate of quinine. These drugs may be given along with Diet II or I or V. I have been able to *demonstrate clearly* the distinct effect of sodium salicylate and of aspirin in diminishing the glycosuria in certain cases of the milder forms of diabetes, and also in cases of medium severity. Some patients take sodium salicylate better, others aspirin.

The dose of these drugs has usually to be increased to 15 gr. (or more) four times a day before definite effects are obtained, and a careful watch should be kept for toxic symptoms. It is better to use the *natural* salicylate. In certain cases salicylate of quinine, gr. 3 or 4 or more, three or four times a day, has appeared to be of distinct service, though I have not been able to clearly demonstrate this by careful hospital observations as in the case of the two drugs just named.

VII. Fast days are sometimes of service. On these days the patient fasts, and takes only tea or coffee, beef-tea or whisky-and-soda several times and no other food. In the less severe cases the glycosuria ceases on these days, but it often returns in a day or two. It may, however, not return for many days, and occasionally for a considerable time it is much less than before the fast days. But so very often I have found the results of little permanent value. A fast day once a week is useful in some cases, but I much prefer the treatment with the two-hourly fluid Diets III and IV described in this article. I also prefer these diets to the more prolonged fasting which is being so frequently carried out.

When the patient suffers from insomnia or marked neurasthenia, bromide of potassium (gr. 30 at night, or 30 early in the evening and 30 late at night) is suitable. Overwork, mental strain, and worry often increase the glycosuria, and should be avoided as much as possible.

If none of these diets or treatments check the glycosuria, then we must be content if we can diminish it by the various treatments described. After a time it is well to allow a small amount of white bread or milk, or both, especially if the urine should give a persistent reaction with perchloride of iron.

In some cases the most careful treatment fails; in others it is successful for a shorter or longer period and then fails. But in some cases it is successful for many years, or even to the end of life.

If we have checked the glycosuria by diet or drugs, then Diet II may be allowed, and later Diet I. Still later more white bread is cautiously allowed, and then other starchy carbohydrates, if no glycosuria returns. But it is always advisable permanently to forbid sugar and sweet foods containing sugar. The urine should be watched and the diet made more rigid if glycosuria returns, and less rigid when the glycosuria ceases. In this way it is possible in some cases to keep the glycosuria in check for many years, or occasionally until the patient reaches advanced age and dies of some other disease."

I do not recommend this plan for use in the East. It does not give the proteid and carbohydrate values in the precise manner of the Allen Method, and the patent preparations indicated are either impossible to secure, or are beyond the means of the average patient.

*Other dietetic methods of treatment.*—Before the introduction of the Allen method with its principle of carbohydrate re-education, diabetes was treated by the restriction of various articles of diet. Perhaps the greatest exponent of this method, and certainly one of the most scientific, in Von Noorden. In his book, New Aspects of Diabetes, 1912, he gives a comprehensive review of the pathology and treatment of diabetes as it existed at that date. The difference between the two schools is clearly marked by the following extract (Von Noorden, p. 75). "The general direction that the intake of carbohydrates should be restricted or excluded, stands to-day in the foreground of diabetic therapy, just as it did in the previous century. *It is perhaps more emphasized than heretofore !*"

This is absolutely contradictory to the principles of the Allen Treatment, where one finds the co-efficient of carbohydrate after fasting, and attempts constantly to increase it. In Von Noorden's scheme, the carbohydrate is much diminished or excluded, and the necessary energy given by fat; thus, the wasteful—non-utilisable carbohydrate is eliminated and replaced by fat, from which heat and energy are produced. The patient remains in a fair state of nutrition or even increases his body weight. This seems to be the limit of the practitioner's ambition, and the patient is advised that it is better to have a small amount of sugar in the urine and keep up the (apparent) general nutrition than to diminish further the intake of carbohydrate.

In order to carry out this method of treatment with any success, it is necessary to determine the carbohydrate tolerance, for unless this is known we have no idea of the personal factor of each individual patient. The preliminary methods resemble those of the Allen Method. The patient is given his ordinary diet for some days, the urine is measured, and the sugar determined in part of a mixed 24 hours sample. He is next placed on a carbohydrate free diet, plus one hundred grams of white bread (*i. e.*, 60 grams carbohydrate). This is continued for three days. As before, the urine is collected, measured and analysed.

*In very mild cases.*—The urine may become sugar free before the third day, in *medium cases* the sugar will fall to some fifteen grains, whilst in *severe cases* the sugar may equal or even exceed the carbohydrate intake, shewing that the body is forming sugar from protein or other substances.

In such circumstances the strict or carbohydrate free diet is continued, and with it is given a constantly diminishing amount of white bread. Thus, strict diet plus 60 grams white bread is given for two days; next, strict diet with 30 grams white bread for two more days, succeeding this, the diet without carbohydrate is given for four or five days. In all but the most severe cases, the urine becomes sugar free; if a small quantity of sugar still remain, it can sometimes be got rid of by a day's fast or by a vegetable day.

In severest cases, however, there will still be sugar excretion, the sugar being formed from the body proteids or from the proteid of the food. By cautious restricted diet over a long period, diminishing also the proteids and fats, and by interpolating occasional vegetable and fast days, or of oatmeal periods, the patient's urine may sometimes be rendered sugar free.

In the milder and moderate cases, after the urine has been free from sugar for three or four days, twenty grams of white bread may be added every other day until sugar reappears.

" The tolerance of the patient in terms of white bread —or in terms of pure carbohydrate (one gram white bread equals ·6 gram carbohydrate) has thus been determined."

While testing the carbohydrate tolerance, sodium bicarbonate should in every case be given when the carbohydrate tolerance falls below sixty grams. In mild cases twenty grams should be given daily, in severer cases forty grams.

The following table of 'strict,' standard, or carbohydrate free diet (together with most of the above material) is taken from Barker's excellent article in Monographic Medicine, p. 826. The diet is not really carbohydrate free, as most of the vegetables contain 4—6 per cent. of carbohydrate and the meat contains some glycogen.

1. *Meat.* Including fowl, fish, oysters, sausages, sweetbreads, kidney, brain, but not liver, sauces containing flour are excluded. Cooked meat contains twenty to thirty per cent. protein ; cooked fish contains twenty per cent. protein.

2. *Eggs.* 1 English hen's egg weighing fifty grams equals six grams protein plus five grams fat, or seventy-one calories ; the protein content of one egg is the equivalent of that of twenty grams cooked fish.

3. *Cheese.* Usually contains 25 per cent. of protein and from 12 to 75 per cent. of fat.

4. *Fats.* Butter (85 to 99 per cent. fat) ; olive oil (100 per cent. fat) ; fat bacon (90 to 95 per cent. fat) ; bone marrow, ghi and mustard oil.

5. *Certain vegetables.* Spinach, cabbage, cauliflower, asparagus, young rhubarb, French beans, artichokes, cucumbers, mushrooms ; these vegetables can have much butter added, but no sugar ; they contain four to six per cent. of carbohydrate. By boiling twice or thrice in different waters they can be rendered almost carbohydrate free.

6. *Certain salads.* Lettuce, endive, cress, cucumber, tomato, French beans, asparagus, olives, oil and vinegar may be added, as well as vinegar and pepper.

7. *Certain fruits and nuts.* Young gooseberries, cranberries, currants, almonds, walnuts.

8. *Certain soups.* Made without flour, though parmesan cheese, roberant or gliadin may be used for thickening; thus consommé or bouillon may contain egg, cut green vegetables, asparagus tips, meat cubes, etc.

9. *Drinks.* Lemonade, unsweetened mineral waters, coffee or tea (with saccharin, saxin, or hediosite to sweeten); Moselle wine, Rhine wine, claret, sherry, whisky or brandy.

ACCESSORY "CARBOHYDRATE CONTAINING" DIET (TO BE USED IN PRESCRIBED AMOUNTS ONLY).

| | Carbohydrate content percentage | Amount in grams equal to 20 grams white bread or 12 grams carbohydrate. |
|---|---|---|
| White bread ... | 60 per cent. | 20 |
| Zweiback ... | 70 ,, | 17 |
| Oatmeal ... | 67 ,, | 18 |
| Rice or macaroni ... | 70–80 ,, | 15–17 |
| Cocoa ... ... | 30 ,, | 40 |
| Potato ... ... | 18–20 ,, | 60–70 |
| Turnip ... ... | 7–10 ,, | 120–170 |
| Jerusalem artichoke... | 15–20 ,, | 60–80 |
| Grapes, plums, peaches, apples ... | 10 ,, | 120 |
| Melons, strawberries | 5 ,, | 240 |
| Milk ... ... | 4–8 ,, | 250 |
| Thick cream ... | 3 ,, | 400 |
| Beer ... ... | 4–5 ,, | 240–300 |
| Oranges ... | 6 ,, | 200 |

Thus, if a patient were allowed, say, 80 grams of white bread (48 grams carbohydrate), he might substitute for it.

(1) 30 grams white bread + half a litre milk.
(2) 30 grams white bread + 150 grams potato.
(3) 30 grams rice + half a litre milk.

*Fast Day* (Naunyn).—Only water, bouillon, lemonade and wine or whisky (100 c.c.).

*Vegetable Day* (Von Noorden).—Three meals are given, each consisting of 250 grams thrice cooked vegetables and salads (from the strict list); to these are added in the 24 hours four to six eggs, one hundred grams or more of butter, a little fat bacon, the yolks of three or four eggs, coffee, tea, lemonade, bouillon and a pint of wine. Two or three such days may follow one another if desired.

*Tolerance for particular carbohydrates.*—In diabetes cases, the special tolerance and the special sensitiveness for particular carbohydrates, particular proteins, and particular fats may have to be tested. Some patients are protein sensitive, doing better on a low protein ration; others are particularly sensitive to certain forms of carbohydrate, while they are fairly tolerant of others. Hence we have had the various carbohydrate cures, including the rice cure, the wheat cure, the potato cure, the rye cure, and most important of all, the oatmeal cure of Von Noorden.

*The Oatmeal cure.*—In severe cases of diabetes mellitus with high acidosis, as well as in cases of medium severity, the oatmeal cure is often well tolerated, and, properly applied may lead to an increase in the carbohydrate tolerance of the patient. In principle, it consists in giving a large amount of carbohydrate in the form of oatmeal, along with a large fat ration and a small protein ration.

Thus in 24 hours the patient receives—

1. 250 grams oatmeal or rolled oats (cooked slowly for 2 or 3 hours).
2. 200 to 300 grams butter (or butter and bacon).
3. 4 to 6 eggs (or 50 to 60 grams vegetable protein, such as gliadin or roberant).
4. 100 to 150 grams green vegetables (from the strict list) or salad.
5. Some alcohol (a pint of hock or 100 c.c. whisky and water).
6. Coffee, tea, lemonade, mineral water.

Before testing the patient's response to such an oatmeal cure, it is important to prepare him by several, days of strict diet, and one or two "vegetable days" in order to reduce the glycosuria, and hyperglycosuria. He is then given from two to four "oatmeal days," followed by one or two "vegetable days;" after this a few days of "standard diet" are permitted. When well borne, two, three, or even more of such cycles may be allowed. In many cases, acidosis diminishes markedly and tolerance improves greatly. In some of the grave cases, no benefit results; the sugar output remains high (120 to 150 grams) the acidosis is not lessened, nor does the general condition improve; here the outlook is grave, though even in such cases, remarkably good results can be obtained by Allen's method. One cannot prophesy beforehand how a patient will respond; the trial must be made to find out. "A satisfactory explanation of the mode of action of the oatmeal cure is still lacking."

The above is Barker's presentation of the dietetic treatment of diabetes before the introduction of Allen's method. Like most other clinicians he has now adopted Allen's method, and with satisfactory results.

It is only in very rare cases that one could persuade an Indian patient to submit to such a course of treatment. The method is so slow, the results so uncertain, the dietetic difficulties so great, that very few Indian patients will go through with it. With Allen's treatment, on the other hand, there is the initial forty-eight hours' fast, and then the complete absence of sugar. That is something very definite accomplished and something to stir the patient's enthusiasm and gain his intelligent co-operation. Great as are the difficulties in treating strict vegetarians by Allen's methods, they are less than by any other, and the success is greater. The more experience I have in using this method, the more convinced I am that it is the method of election for use in India.

# CHAPTER VI.

## GENERAL HYGIENIC MEASURES, TREATMENT BY DRUGS, AND TREATMENT OF COMPLICATIONS.

IN diabetes, as in most other chronic diseases, common sense hygienic measures are of great value. If the opportunity occur, it is advisable to test the sugar and carbohydrate tolerance of other members of the diabetic's family. Should one or more of these people be unable to eat pure sugar without glucose occurring in the urine, early *prophylactic* measures, such as an occasional fast may be tried to avert the disease.

Once the disease is established personal hygiene is of the greatest importance; the skin must be particularly attended to, for the susceptibility of the diabetic to boils and carbuncles is well known. Hence the importance of the daily bath, the most scrupulous cleanliness and the instant attention to any small boil or pimple. The liniment of iodine promptly applied is most useful in these cases, and a lead and opium lotion will often cut short an irritating pruritus. In Indian patients who have the armpits shaved, special care should be taken, for boils in that situation are frequent. Gentle massage, either dry, or with oil after the Indian fashion, is very useful, and so, too, is the Turkish or vapour bath. Exercise must be carefully regulated, it must be sufficient but not excessive.

A moderate amount helps to burn up carbohydrate, but an excessive amount, in severe cases, favours the occurrence of coma.

Another important matter is the prevention of constipation and the care of the digestion in general. Constipation, we know, is very common in diabetes, and sometimes is most obstinate; even when stools are passed they are apt to be offensive and contain undigested matter. A morning saline is almost a necessity in these cases, and it may be usefully combined with occasional fractional doses of calomel, or with medicinal paraffin or cascara for daily use.

Another useful remedy is the B. bulgaricus; if an active preparation be secured, and the patient be able to deal with a few ounces of milk, the use of this bacillus will often stop intestinal fermentation and may even stimulate the pancreas to greater activity when the gastric acidity is low.

Special attention, too, should be given to the teeth. The gums are apt to recede, pyorrhœa is common, and the teeth may even fall out. Iodine locally is of the greatest use here, and a course of emetine should be given for the pyorrhœa; a few injections, say half a dozen, will usually make a marked difference in this respect.

The avoidance of these apparently minor complications or hygienic errors is of the greatest importance in preserving the health and life of the patient. An old prescription for the attainment of longevity is to "acquire a chronic disease and look after it" and of no disease in this more true than of diabetes.

*Drug treatment in diabetes.*—The list of drugs that have been advocated for diabetes is a very long one, and its very length shows how ineffectual they are. The one drug that I have found of use is opium. It certainly allows the patient to deal with a larger quantity of carbohydrate, probably because it delays the digestive processes and slows the rate at which carbohydrates are absorbed. It also soothes the nervous system and diminishes nervous excitability.

It has a traditional use in the East and is widely used. Fortunately, diabetics seem to be able to deal with large doses without ill-effects. The ordinary crude opium may be used in a commencing dose of one grain daily, with a daily increment of one grain until the desired effect is produced. Fifteen and sixteen grains daily may be given without producing narcotism should the symptoms demand it ; this dose, although large, is of course much less than that taken by many habitual users of the drug. With the Allen treatment it is not often necessary, but it may be useful in quietening the patient, and rendering him more amenable to treatment.

The alkaloids of opium are sometimes used ; morphia is used by some physicians, but the most popular alkaloid is codeina. The sulphate of codeina is prescribed in half grain doses, gradually increased to five grains daily. It is fashionable, but is expensive, and has no advantages over the crude opium.

Arsenic, too, has been vaunted as a specific. It may be given as liquor arsenicalis or as arsenious acid, pushing it till the limit of toleration is produced. I have seen no benefit from its use.

Aceto-salicylic acid, phenacetin, and antipyrine have all been tried, generally without success. Their action in producing a fallacious di-acetic acid reaction to ferric chloride should not be forgotten.

Bromides, salicylates, and iodides have all been used, and uranium nitrate has had a vogue. Whilst the bromides are useful in soothing the nervous system, and the iodides may be useful where syphilis is suspected, as remedies for diabetes they are useless.

Mineral waters and spas are of some value, the latter chiefly because the patient will the more readily submit to the regimen of treatment at a spa than he will at his own home.

Contrexeville and Vichy water, Carlsbad salts in the morning, are alkaline and mildly laxative, and thus aid the general hygiene of the patient.

For the neuritis, ionic medication with a 2 per cent. solution of potassium iodide is of great use. The current should be a weak one at first—say 3 milliamperes, then cautiously increased. Treatment is given every other day.

## SURGICAL OPERATIONS IN DIABETICS.

Sugar in the urine is rightly considered as a contraindication to surgical operations, and no one would willingly operate when glycosuria had been detected. Whilst this is true for æsthetic operations such as hydrocele, hernia, simple tumours, and so on, there are occasions when the risk of surgical interference must be faced. It may be evident that the risk from the surgical ailment is less than the risk of the coma which may be induced by the anæsthetic.

Should the condition not be very urgent, such as a carbuncle, or an impending gangrene, the patient should first be rendered sugar-free by Allen's method and then operated on.

If possible, the patient should be rendered free from diacetic or oxybutyric acids before operation, for, as previously demonstrated, these substances are the precursors of diabetic coma.

Wherever practicable, spinal analgesia with stovaine should be used in place of a general anæsthetic, and thus all areas up to the umbilicus can be easily dealt with.

In cases of gangrene of the lower extremity, the amputation must be performed high up, certainly not lower than the knee-joint and preferably through the thigh. The operation should be performed as rapidly as possible and every effort made to combat shock.

It is to be noted that, occasionally, after a serious surgical operation, sugar may disappear from the urine and remain absent.

Miller, Joslin, and Manges report cases of hysterectomy for malignant disease, and for uterine febroids and two cases of prostatectomy. All of these were diabetics; they recovered from their operations and remained sugar-free in spite of a liberal diet.

Reference has already been made to the occasional disappearance of sugar when tuberculosis appears, or when interstitial nephritis with arterio-sclerosis occurs.

*Boils and Carbuncles.*—These are most annoying and sometimes dangerous complaints. The first indication is to get the urine sugar-free and then to make free incisions. Following this, Kocher's suction method may be used, or, where practicable, baths of hypertonic saline may be used for three hours daily. A vaccine may be prepared from micro-organisms detected, but its use should be deferred until the urine has been rendered sugar-free.

*The Treatment of Diabetic Coma.*—Coma no longer represents the culmination of diabetes, it is an avoidable accident. That should be the principle guiding every physician who has continuous charge of a diabetic. But there are certain precautions that should be observed to ward off coma, and there are certain methods of treatment that may be adopted should this complication occur. Constipation must be particularly guarded against, also excessive exertion, physical or mental. It was formerly held that absence of carbohydrate, restriction of diet, and so on might bring on coma. In the vast majority of cases this is untrue. Absolute starvation, following the Allen plan, causes the disappearance of sugar and of the acidosis. In a very few "severe" cases there may be an attack of vomiting, followed by increasing acidosis, coma, and death, all commencing from the time that alimentary rest begins, but these cases are extremely rare, and probably only occur in cases that are on the verge of coma when treatment begins.

The importance of acidosis should never be overlooked, and therefore tests for diacetic acid should be regularly made, as regularly as the daily test for sugar. Should this test be positive, and remain positive, alcohol in divided doses should be administered, all fats should be eliminated from the diet, and bicarbonate of soda administered in large doses. The carbohydrate intake should be steadily increased, particularly in the form of milk, as sometimes when this is done the acidosis disappears. If need be, some sugar must be allowed to appear in the urine, for the outlook in coma is so grave that everything must be sacrificed to avert it.

The soda, either as carbonate or bicarbonate, preferably the latter, should be given in large doses—thirty to forty grains thrice daily, in the hope of stopping the acid intoxication.

The alkali neutralises the acid in the circulatory blood, and the tissue bases (sodium, potassium, etc.), together with the ammonia derived from the breaking down of the body proteids, are spared.

Should coma once occur, the outlook is extremely grave, and recovery is extremely rare. The general lines of treatment, elimination of fat and administration of carbohydrate, have already been indicated. The lævulose of milk is the carbohydrate most easily assimilated, and, in addition to giving large quantities by the mouth, Van Noorden has advocated the intravenous injection of 5 to 10 per cent. solution of lævulose.

Undoubtedly, the administration of large quantities of sodium bicarbonate by intravenous injection, and by the rectum, will *temporarily* relieve a certain proportion of coma cases. The quantity of soda given is sometimes enormous.

Should swallowing still be possible, one to two drams every hour may be given, and double this quantity *per rectum.*

Intravenous injections are generally more effective. Quantities varying from 20 to 80 grams of bicarbonate of soda have been given at one injection, or a 3 to 5 per cent.

solution of sodium carbonate. Sometimes quite striking results are obtained, though only temporarily. The respiration becomes much quieter, and occasionally there may be a short return of consciousness; this may even be sufficient to enable the patient to speak, sign his name to documents and so on. But, almost invariably, there is a recurrence of the coma and death within thirty-six hours.

In the statistics quoted on page 68 it was shown that about half the cases of diabetes died from coma. Joslin, as the result of twelve months' experience of the Allen treatment, says that no patient during this period, whom he has taught to examine his own urine, has died, and this, we may hope, will be the general result, so far as coma is concerned, when the newer and more scientific methods are more widely applied.

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